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THE VALUE OF THE HIPPURIC ACID TEST AND TAKATA-ARA REACTION IN THE INVESTIGATION OF HEPATIC DEFICIENCY.[1]

By MARGARET HENDERSON[2] and BERYL SPLATT.

(From the Walter and Eliza Hall Institute, Melbourne.)

The Hippuric Acid Excretion Test.

FOLLOWING earlier work of his own[(1)] on the conjugation of benzoic acid in the dog and other animals, Quick[(2)] in 1933 suggested that this process might be used as a test of liver function in man. The test is based on the assumption that benzoic acid is conjugated with glycine in the liver. It is probable that two processes are involved: the synthesis of glycine and a conjugating mechanism dependent on an enzyme. According to later work of Quick,[(3)] this enzyme is present mainly in the liver, the small amount in the kidneys being insufficient to compensate for liver damage. Quick[(3)] also demonstrated that in man the administration of extra glycine could increase the amount of benzoic acid conjugated to hippuric acid. Probstein and Sol Londe[(4)] found that the administration of glycine to patients with liver damage increased the excretion of hippuric acid, but not to normal levels; this confirmed the suggestion that some other mechanism was involved. The clinical studies published by Quick,[(3)(5)] Boyce and McFetridge,[(6)] Bartels,[(7)] Probstein and Sol Londe[(4)] and De Lor and Reinhart[(8)] indicate that the test has found many useful clinical applications.

The method used in this series is that originally described by Quick.[(3)] The patient is allowed to have a light breakfast. The bladder is emptied and the patient is given by mouth six grammes of sodium benzoate dissolved in about two ounces of water. The bladder is emptied one, two, three and four hours later. If the patient cannot pass urine at intervals of one hour, the bladder should be emptied at the end of four hours and the hippuric acid estimated in the single specimen after concentration by evaporation. Occasionally a patient complains of nausea, but this seldom occurs if he is lying down. One or two cups of fluid may be given if desired during the period of the test.

The volume of each specimen of urine is measured and the urine is acidified with concentrated hydrochloric acid, Congo red paper being used as an indicator. Crystals of hippuric acid are precipitated and allowed to form overnight. The specimens are then filtered and the crystals washed free from hydrochloric acid with cold distilled water, dissolved in hot water and titrated with normal sodium hydroxide solution. One cubic centimetre of normal sodium hydroxide solution is equal to 0·18 gramme of hippuric acid, and each 100 cubic centimetres of urine at 15° C. can dissolve 0·33 gramme of hippuric acid, so the total amount of hippuric acid in any specimen is given by the following calculation: (No. of c.c. of N.NaOH × 0·18) + $\frac{\text{(Volume of urine in c.c.} \times 0{\cdot}33)}{100}$. The result is expressed as the number of grammes of benzoic acid excreted in four hours, and to obtain this figure the number of grammes of hippuric acid excreted is multiplied by 0·68. Confusion has arisen in the literature, some authors speaking of the number of grammes of hippuric

[1] This work was aided by a grant from the National Health and Medical Research Council.

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acid excreted, when apparently the result is being expressed as benzoic acid. We have followed Quick's original paper and converted the hippuric acid excreted to benzoic acid and expressed the results as the number of grammes of benzoic acid excreted in four hours. If no precipitate is found in the urine, or if the volume of urine passed is over 150 cubic centimetres, the urine should be evaporated until a precipitate of hippuric acid is obtained after acidulation. If no precipitation occurs, as in some cases in which function is poor, the result can only show that not more than a certain amount of hippuric acid was excreted. We have not used the modification suggested by various authors, such as Weichselbaum and Probstein,[5] who advocate decreasing the solubility of hippuric acid in urine by the addition of sodium chloride (30 grammes per 100 cubic centimetres of urine), and who have described useful modifications for bile-stained and protein-containing urine. Quick[3] has recommended the use of ammonium sulphate (5 grammes per 10 cubic centimetres of urine) to decrease the solubility of hippuric acid and has also modified the test for intravenous use.

The present investigation is a study of the effectiveness of the hippuric acid excretion as a test of liver function. It is based on tests carried out on 190 adult subjects, of whom 89 were suffering from disease of the liver and biliary tract and 86 from various other disorders, and 25 were normal adults or patients convalescent from minor surgical conditions.

Normal Excretion.

The amount of three grammes of benzoic acid is regarded by Quick[3][4] as the average normal quantity excreted; less than 90% of this amount (under 2·7 grammes) is considered abnormal. Among the 25 controls listed in Table I we found the average amount excreted to be 3·6 grammes. The figures for men and women did not differ significantly (3·6 grammes for 16 men and 3·5 grammes for 9 women); but the average figure for the eight normal volunteers was 4·0 grammes, as against 3·3 grammes for the 17 hospital patients. Three hospital patients without liver disease excreted less than three grammes.

TABLE I.
Controls.

Case Number.	Sex of Patient.	Age. (Years.)	Clinical Condition.	Grammes of Benzoic Acid Excreted in Four Hours.
I	M.	25	Normal.	4·3
II	M.	25	Normal.	3·5
III	M.	24	Normal.	4·5
IV	M.		Normal.	3·6
V	M.	29	*Pes cavus.*	3·4
VI	M.	22	Epilepsy (post-traumatic).	3·7
VII	M.	49	Epilepsy (idiopathic).	3·4
VIII	M.	36	Convalescent pneumonia.	3·7
IX	M.	50	Convalescent pyelitis.	3·1
X	M.	17	Fractured femur.	2·1
XI	M.	30	*Fistula in ano.*	3·7
XII	M.	22	Ulcer of leg (post-traumatic).	3·8
XIII	M.	30	Fractured tibia.	3·9
XIV	M.	46	Varicose ulcer.	2·9
XV	M.	51	Cellulitis (convalescent).	3·1
XVI	M.	30	Plumbism (convalescent).	3·4
XVII	F.	35	Normal.	3·6
XVIII	F.	25	Normal.	3·7
XIX	F.	19	Normal.	3·8
XX	F.	24	Normal.	4·3
XXI	F.	20	Fractured femur.	3·2
XXII	F.	23	Fractured humerus.	2·7 2·6
XXIII	F.	18	Fractured femur.	4·1
XXIV	F.	17	Perianal abscess.	3·1
XXV	F.	21	Perianal abscess.	2·8

Probstein and Sol Londe[6] suggest a higher figure for the normal than Quick's; but their figure is based only on the results obtained from healthy volunteers. They also suggest using a dose of four grammes, of which two grammes should be eliminated in two hours, as they consider that the maximum output should be attained in the second hour, and that this is a good index of the liver function. Our figures do not support this, as among normal patients the maximum amount excreted per hour occurred in any of the four specimens, and after the ingestion of six grammes the amount excreted after two hours was often less than two grammes when the final result was normal. The figure of three grammes seems to allow for what are undoubtedly wide variations in the normal subject.

Hepatic Disease.

In 65 cases the incidence of disease was predominantly in the liver (Table II). Of six cases of catarrhal jaundice, the excretion of benzoic acid was diminished in five; but in only one case was the diminution pronounced (Case III). This patient had a severe grade of jaundice with an enlarged spleen. In two cases in which the tests were repeated after medical treatment, an improvement in the result was observed. Of nine cases of toxic hepatitis, four were due to arsenical preparations. In these four cases diminished excretion of benzoic acid was found when the first test was made. The benzoic acid excretion of these three patients rapidly improved to normal after two weeks of medical treatment; the fourth patient was transferred to another hospital. In the remaining five cases of toxic hepatitis there were two deaths (Cases IX and X); autopsy revealed extensive liver damage. Both of these patients had grossly impaired excretion and failed to respond to medical treatment.

In all the eight cases of syphilis of the liver the initial tests revealed a very poor excretion (0·6 to 1·8 grammes). Death occurred in one case (Case XVII), and autopsy revealed a typical large, scarred liver with stones in the common bile duct and cholangitis. In two cases the improvement that followed antisyphilitic treatment could be measured by the increase in the amount of benzoic acid excreted.

Tests were performed on twenty patients with cirrhosis of the liver, six of whom gave a history of alcoholism. Ascites was present in thirteen cases and liver enlargement in nine. In the eleven cases which came to autopsy the amount of benzoic acid excreted lay between 0·7 gramme and 1·6 grammes and in the remaining nine cases between 1·2 grammes and 2·4 grammes. There was no correlation between the excretion of benzoic acid and the number of units of serum bilirubin present.

Case XXIX is of interest.

The patient was seen in 1937, when she was suffering from alcoholic peripheral neuritis; the amount of benzoic acid excreted was then 2·8 grammes. In the next two years she developed ascites and liver enlargement and the amount excreted dropped to 2·3 grammes. Laparotomy revealed a large nodule on the liver.

In Case XXXI cirrhosis resulted from subacute yellow atrophy of the liver following a septic abortion.

Two patients with hepato-lienal fibrosis were observed (Cases XLV and XLVI); both showed diminished excretion. One was readmitted to hospital twelve months later, deeply jaundiced, and showed further impairment; the other underwent splenectomy and has been fairly well, the result of the hippuric acid test being now normal.

In four cases a diagnosis of hæmochromatosis was made. In two of these the excretion was within normal limits. In Case XLVIII slight impairment was present and in Case XLVII gross impairment. This patient also presented a diminished plasma albumin content with ascites and pleural effusion and was the only one of the four with jaundice.

Five patients with hydatid disease of the liver were tested. In Case LI the calcified mass was discovered during X-ray examination; there was no liver enlargement and the benzoic acid excretion was satisfactory. In the other four cases in which the patients came to operation the excretion was impaired; in three out of the four it became normal within a few weeks after operation. If the ability of the liver to hypertrophy in cases of hydatid infestation

TABLE II.

Diseases of the Liver.

Case Number.	Patient's Sex and Age. (Years.)	Diagnosis.	Van den Bergh Reaction.		Takata-Ara Reaction.	Grammes of Benzoic Acid Excreted in Four Hours.	Clinical Outcome.	Operative or Autopsy Findings.
			Qualitative.	Quantitative.				
I	M. 18	Catarrhal jaundice.	Biphasic.	6·5		4·3	Recovery.	
II	M. 27	Catarrhal jaundice.	Biphasic.	8·5		2·7	Recovery.	
III	M. 30	Catarrhal jaundice.	Biphasic.	10·5		1·0 (18/8/36) 1·3 (23/8/36)	Recovery.	
IV	M. 30	Catarrhal jaundice.	Immediate.	11·0		2·2	Recovery.	
V	M. 38	Catarrhal jaundice.				2·7	Recovery.	
VI	M. 32	Catarrhal jaundice.	Biphasic.	6·0	(i) Strong. (ii) Moderate. (iii) Moderate.	2·8 (6/9/41) 2·1 (10/9/41) 3·6 (22/10/41)	Recovery.	
VII	M. 55	Hepatitis, chronic cholecystitis.	Biphasic.	8·5		1·9	Cholecystectomy; no improvement.	
VIII	F. 51	Toxic hepatitis.	(i) Immediate. (ii) Delayed.	8·0 2·5		1·7 (22/11/38) 2·1 (16/12/38) 2·3 (23/1/39)	Recovery.	
IX	F. 43	Toxic hepatitis.	Delayed.	5·0	Strong.	1·2 (13/12/39) 1·2 (28/12/39)	Death two months later.	Necrosis of liver. Liver large, scarred, bright yellow, large areas of atrophy. Some cellular fibrous tissue reaction.
X	M. 39	Toxic hepatitis.	Biphasic.	7·0		<1·4	Laparotomy; death.	Toxic hepatitis. Liver enlarged and bile-stained.
XI	M. 51	Toxic hepatitis.	Delayed.	3·0		2·2	Improvement.	
XII	M. 35	Arsenical hepatitis.	Biphasic.	10·5		<0·5		
XIII	M. 29	Arsenical hepatitis.	Immediate.	8·0		1·7 (28/8/40) 3·3 (7/9/40)	Rapid improvement.	
XIV	F. 49	Arsenical hepatitis.	Immediate.	9·0		2·2 (22/4/41) 3·4 (30/4/41) 3·6 (14/6/41)	Rapid improvement.	
XV	M. 34	Arsenical hepatitis.	Immediate.	12·0	No reaction.	0·7 (17/7/41) 1·7 (24/7/41) 2·9 (31/7/41)	Recovery.	
XVI	M. 76	Syphilitic hepatitis.	Immediate.	10·0		0·9 (4/8/36) 2·3 (26/8/36) 3·2 (22/9/36)	Improved on treatment.	
XVII	M. 72	Visceral syphilis; stone in common bile duct.	Biphasic.	8·5		0·8	Laparotomy; death.	Cholangitis. Liver large, greenish, friable, with irregular deep scars.
XVIII	M. 59	Syphilitic hepatitis.	Delayed.	6·5		1·8	Improved.	
XIX	M. 47	Syphilitic hepatitis.	Biphasic.	8·0	Moderate.	0·8		
XX	F. 44	Syphilitic hepatitis.	Immediate.	12·0	Strong.	0·8 (7/12/39) 1·8 (8/1/40)	Improved.	
XXI	M. 51	Syphilitic hepatitis; *tabes dorsalis*.			No reaction.	2·0 (22/2/40) 0·6		
XXII	M. 57	Syphilitic hepatitis; diabetes.	Delayed.	4·0	Strong.	1·1	Laparotomy.	Cirrhosis of liver.
XXIII	M. 55	Cirrhosis (probably syphilitic).	Biphasic.	8·0	Strong.	1·0		
XXIV	M. 51	Cirrhosis of liver (? alcoholic).	Delayed.	3·0		1·1	Death.	Liver large and leathery. Intense cirrhosis of portal type. Slight regeneration.
XXV	F. 53	Cirrhosis of liver; pyelitis.	Biphasic.	6·5		1·6	Death nine months later.	Liver small, greenish-yellow with periportal fibrosis. Multilobular cirrhosis.
XXVI	M. 71	Cirrhosis of liver.				0·7	Death.	Liver shrunken, brownish gross multilobular cirrhosis.
XXVII	M. 56	Cirrhosis of liver; cardiac failure.	Biphasic.	7·0		1·3	Death two years later.	Liver small, yellow, fatty, scirrhotic. Pulmonary abscess.
XXVII	F. 48	Cirrhosis of liver (? alcoholic). Hæmatemesis.	No reaction.			2·0	Recovery.	
XXIX	F. 39	(i) 26/6/37: alcoholic peripheral neuritis. (ii) 12/5/39: cirrhosis of liver.	Delayed.	3·0		2·8 (26/6/37) 2·3 (12/5/39)	Developed ascites and liver enlargement.	
XXX	F. 17	Cirrhosis of liver.	Biphasic.	6·0		1·3 (9/2/39)	Recurrence of jaundice, 1940.	Liver very enlarged with hard nodules. Microscopic findings: Marked cirrhosis with fibrosis and cell accumulation.
			Biphasic.	5·0		1·2 (6/3/39)	Laparotomy.	
XXXI	F. 34	Cirrhosis of liver (seven months after subacute yellow atrophy).	Biphasic.	4·6		1·5	Death.	Liver small and rounded, with yellow nodules of regenerating tissue. Dense portal fibrosis, lymphocytic infiltrations.
XXXII	F. 31	Cirrhosis of liver.	Biphasic.	8·5		<1·4	Laparotomy; death.	Liver very large, greenish, tough. Intense portal cirrhosis, large area of atrophy, little regeneration. Spleen enlarged.
XXXIII	M. 46	Cirrhosis of liver.	Delayed.	4·0		1·1	Death.	Liver small, yellow-grey. Intense portal cirrhosis.
XXXIV	F. 39	Cirrhosis of liver.			Moderate.	1·1	Death.	Liver large, yellowish, granular. Intense portal cirrhosis. Infected ascites.
XXXV	F. 49	Cirrhosis of liver (? alcoholic); mild hæmatemesis.	Delayed.	3·0	No reaction.	2·2	Temporary improvement.	

TABLE II.—*Continued.*

Diseases of the Liver.—Continued.

Case Number.	Patient's Sex and Age. (Years.)	Diagnosis.	Van den Bergh Reaction.		Takata-Ara Reaction.	Grammes of Benzoic Acid Excreted in Four Hours.	Clinical Outcome.	Operative or Auto. Findings.
			Qualitative.	Quantitative.				
XXXVI	M. 58	Cirrhosis of liver.	Delayed.	3·0		1·1	Laparotomy; death.	Liver small, multilobular cirrhosis. Ascites.
XXXVII	F. 63	Cirrhosis of liver; hypertension.	No reaction.			1·5	Death.	Liver small, yellow, coarsely nodular. Marked portal cirrhosis without regeneration.
XXXVIII	M. 50	Cirrhosis of liver.	Biphasic.	6·0		<1·2	Omentopexy; death.	Liver very hard nodular; cirrhosis.
XXXIX	M. 64	Cirrhosis of liver.	No reaction.		Weak.	1·8	Under observation.	
XL	M. 65	Cirrhosis of liver (? alcoholic).			Moderate.	1·2	Under observation.	
XLI	M. 47	Cirrhosis of liver (? alcoholic). Hæmatemesis.	Delayed.		No reaction.	2·4	Improved.	
XLII	F. 56	Cirrhosis of liver.	Delayed.	3·0		1·4		
XLIII	F. 56	Cirrhosis of liver.				1·6		
XLIV	F. 19	? Subacute yellow atrophy.	Delayed.	4·0		1·5		
XLV	F. 54	Hepato-lienal fibrosis.	Biphasic. (1940).	7·0	Moderate.	2·1 (1940)	Under observation.	
			Immediate (1941).	12·0	Strong.	1·6 (1941)		
XLVI	F. 46	Hepato-lienal fibrosis.	No reaction.			2·2 (1940)	Splenectomy.	
			Delayed.	2·0	Moderate.	2·9 (1941)		
XLVII	F. 17	Hæmochromatosis (atypical).	Biphasic.	5·5	Strong.	0·9	Temporary improvement.	
XLVIII	M. 61	Hæmochromatosis; diabetes.	No reaction.		Weak.	2·4		
XLIX	M. 46	Hæmochromatosis; diabetes.	No reaction.		No reaction.	2·9		
L	M. 35	? Hæmochromatosis.	No reaction.		No reaction.	3·0		
LI	F. 60	Calcified hydatid cyst of liver.	No reaction.		No reaction.	2·9	Symptomless.	
LII	M. 67	Hydatid cyst of liver.				2·1	Cyst marsupialized.	Liver appeared mildly cirrhotic. Hydatid cysts.
LIII	M. 61	Hydatid cyst of liver.	Before operation; delayed.	2·5	No reaction.	1·5 (before).	Hydatid cysts removed.	Hydatid cysts.
			After operation; delayed.	3·0	No reaction.	3·2 (after).		
LIV	F. 28	Hydatid cyst of liver.				1·6 (before).	Cyst marsupialized.	Hydatid cysts.
						3·4 (after).		
LV	F. 35	Hydatid cyst of liver.			No reaction.	1·7 (before).	Removal of cyst.	Very large hydatid cyst.
					Strong.	3·3 (after).		
LVI	F. 72	Secondary carcinoma in liver.	Immediate.	7·0		1·5	Death.	
LVII	M. 61	Carcinoma of liver.	Biphasic.	6·5		0·3	Death ten days later.	
LVIII	M. 38	Melanotic sarcoma of liver.				2·3		
LIX	M. 57	Carcinoma of ileum; hepatic metastases.	Immediate.	10·0		1·81	Laparotomy.	Secondary deposits in liver and in glands around common bile duct.
LX	F. 49	Carcinoma of adrenal gland; hepatic metastases.[1]				3·1	Death.	Liver congested, friable, studded with tumour nodules. Portal canals clear.
LXI	M. 51	Carcinoma of pancreas; hepatic metastases.	Delayed.	3·0		2·5	Death.	Liver very enlarged, largely replaced by tumour tissue.
LXII	M. 53	Carcinoma of bronchus; hepatic metastases.	No reaction.			0·7	Death.	Extremely large and numerous tumour nodules throughout liver.
LXIII	M. 60	Carcinoma of bronchus; hepatic metastases.	Biphasic.	5·0	Negative.	1·5	Death nine days later.	Liver very large (150 ounces). Numerous tumour nodules and diffuse infiltration of right lobe. Gall-bladder normal.
LXIV	F. 56	Carcinoma of uterus; secondary carcinoma of liver.	Immediate.	10·0	Strong.	1·8	Death six weeks later.	Numerous secondary deposits in liver. Pancreas infiltrated with tumour tissue.
LXV	F. 38	Secondary carcinoma of liver.	Immediate.	8·0		2·1		

[1] Patient not jaundiced.

and the remarkably rapid improvement after operation in the ability to conjugate benzoic acid are taken into account, it appears that the diminished function is probably due to pressure and circulatory interference rather than to replacement of liver substance.

In the ten cases of metastatic carcinoma of the liver the test produced very variable results, as would be expected. The amount excreted ranged from 0·3 gramme to 3·1 grammes of benzoic acid and showed no correlation with the degree of jaundice. From examination of the autopsy findings it does not appear that the impaired function depends entirely either on the amount of liver involved or on the degree of obstruction to the portal canals.

Disease of the Gall-Bladder and Bile Ducts.

The 24 cases of pathological conditions of the gall-bladder and bile ducts include four cases of carcinoma of the pancreas causing common duct obstruction (Table III). Of three patients with chronic cholecystitis without

TABLE III.

Conditions Affecting the Gall-bladder and Bile Ducts.

Case.	Patient's Sex and Age. (Years.)	Diagnosis.	Van den Bergh Reaction.		Takata-Ara Reaction.	Grammes of Benzoic Acid Excreted in Four Hours.	Clinical Outcome.	Operative or Autopsy Findings.
			Qualitative.	Quantitative. (Units.)				
I	F. 55	Chronic cholecystitis following typhoid fever.	Biphasic.			2·5	Improved with medical treatment.	
II	F. 51	Cholecystitis; cholelithiasis.			No reaction.	3·2	Cholecystectomy.	Chronic cholecystitis and cholelithiasis.
III	F. 49	Chronic cholecystitis; coronary sclerosis.	No reaction.		No reaction.	3·2		
IV	F. 41	Biliary dyscrasia.	Delayed.	3·5	No reaction.	2·9	Laparotomy.	Gall-bladder and stomach appeared normal.
V	M. 65	Cholelithiasis; obstructive jaundice; renal stone.	Biphasic.	6·0	Weak.	1·1		
VI	M. 54	Cholelithiasis; transient jaundice.	Immediate.	7·5	No reaction.	3·1	Jaundice cleared rapidly.	
VII	F. 60	Cholelithiasis; obstructive jaundice.	Immediate.	7·0	No reaction.	1·1 (29/5/41)	Improved on medical treatment.	
			Delayed.	3·0		2·8 (8/6/41)		
VIII	F. 65	Cholelithiasis; cirrhosis of liver.				1·0 (11/7/36)	Cholecystectomy.	Cirrhosis of liver.
						2·8 (20/8/36)		
IX	M. 47	Cholelithiasis; obstructive jaundice (three days).	Biphasic.	5·0		4·6	Jaundice cleared.	
X	F. 70	Cholelithiasis; obstructive jaundice.				2·4	Cholecystectomy and recovery.	Stone in common bile duct.
XI	M. 59	Cholelithiasis; obstructive jaundice.	Immediate.	15·0	No reaction.	<0·8	Cholecyst-duodenostomy; death.	Liver small, shrunken, friable, brownish-green. Stone in common duct, suppurative cholangitis.
XII	F. 50	Cholelithiasis; obstructive jaundice.	Immediate.	12·0	No reaction.	2·5 (18/6/41)	Improved on medical treatment.	
			Biphasic.	6·0	No reaction.	2·4 (25/6/41)		
XIII	F. 47	Cholelithiasis; obstructive jaundice.	Immediate.	14·0	No reaction.	2·6 (4/8/41)	Cholecystectomy.	Chronic cholecystitis, cholelithiasis.
						3·0 (26/8/41)		
XIV	F. 53	? Cholelithiasis; obstructive jaundice.				<1·2	Jaundice cleared on medical treatment.	
XV	M. 38	Stricture of common bile duct; intermittent jaundice.	Delayed.	3·0	No reaction.	3·7	Laparotomy and dilatation of duct.	No stones or cirrhosis found.
XVI	F. 62	Stricture of common bile duct.	Immediate.	9·0		1·5		
XVII	F. 49	Stricture of common bile duct.			No reaction.	1·7		
XVIII	F. 29	Occlusion of common bile duct.	Immediate.	10·0		1·6 (3/10/40)	Choledoch-gastrostomy (23/10/40).	
						1·2 (10/11/40)	Dilatation of anastomosis (17/4/41).	
			Immediate.	6·0		1·9 (10/12/40)		
			Immediate.	7·0	Moderate.	2·3 (14/5/41)		
XIX	F. 59	Carcinoma of pancreas; obstructive jaundice.	Immediate.	9·0		0·8 (pre-operative)	Cholecyst-gastrostomy.	Hard nodule in head of pancreas obstructing common bile duct.
						2·5 (post-operative)		
XX	M. 58	Carcinoma of pancreas; obstructive jaundice.	Immediate.	12·0		0·7	Cholecyst-jejunostomy; death.	Carcinoma of head of pancreas. Liver enlarged, green, friable. Suppurative cholangitis. Necrosis and some atrophy of parenchyma.
XXI	F. 70	Carcinoma of pancreas (unproved); obstructive jaundice (3 weeks).	Immediate.	10·0		<1·0		
XXII	F. 80	Carcinoma of pancreas (unproved); obstructive jaundice (6 weeks).	Biphasic.	4·0		0·9 (26/11/38)	Jaundice persistent.	
						1·3 (21/12/38)		
XXIII	F. 67	Carcinoma of bile ducts; obstructive jaundice; *diabetes mellitus*.	Immediate.	13·0	No reaction.	<0·5	Death three weeks later.	Carcinoma of bile ducts with obstruction. Metastases in liver, lymph glands, peritoneum, adrenals and pancreas. Liver very large (72 ounces), deeply bile-stained and contained numerous tumour nodules.
XXIV	M. 68	Carcinoma of gall-bladder.[1]				0·7	Death ten days later.	Carcinoma of gall-bladder with metastases in liver. Liver fairly large (56 ounces), substance red, surface granular. Moderate degree of portal cirrhosis. Portal vein and branches infiltrated with growth and thrombosed. No obstruction to bile ducts.

[1] Patient deeply jaundiced.

jaundice, two had a normal excretion, and one, following a *Bacillus typhosus* infection, some impairment of excretion. In two cases in which jaundice was transitory (Cases VI and IX) excretion was not impaired; in others the results of the test improved after medical treatment (Case VII) or after medical preparation and cholecystectomy (Cases VIII and XIII). Improvement was usually synchronous with a decrease in jaundice. A patient who excreted 0·8 gramme (Case XI) died after operation, and autopsy revealed suppurative cholangitis with extensive degenerative changes in the liver. The diminished excretion in Case V is undoubtedly due in part to poor renal function, which is discussed more fully later. It appears that cholecystitis with or without transient jaundice may not greatly diminish the power of the liver to conjugate sodium benzoate. Quick's[2] findings confirm this. When jaundice is of long duration or when infection of the ducts has supervened, the power of conjugation is definitely impaired. Boyce and McFetridge[1] lay great stress on the value of the hippuric acid test in assessing liver function before operations on the biliary tract; they consider that a series of tests can measure the improvement in the liver function brought about by treatment with glucose and bile salts.

One patient (Case V) with a narrowing of the common duct and a short and rather indefinite medical history had a normal hippuric acid excretion. In three other cases of stricture or occlusion of the duct (Cases XVI, XVII and XVIII) there was definite impairment. Four patients with carcinoma of the pancreas, one with carcinoma of the bile ducts and one with carcinoma of the gall-bladder all excreted less than one gramme of benzoic acid when first tested. Autopsies were carried out in three cases. In a fourth (Case XX), cholecystgastrostomy relieved the jaundice and remarkably improved the hippuric acid excretion.

Renal Disease.

The early, unfounded hypothesis that hippuric acid was synthesized in the kidneys has led to confusion and delay in the acceptance of the test; but it is important to know how renal disease modifies the results. According to Quick,[3] the power of the normal kidney to excrete hippuric acid is 50% greater than the liver's capacity to synthesize it, so that small deviations in kidney function are not likely to be important. However, when there is a failure to excrete nitrogenous products, such as uric acid and urea, hippuric acid will also be retained. In the eleven cases of renal disease in Table IV, either elevated blood urea content or poor urea concentration was present in all. In all these there is some defect in the excretion of hippuric acid; this appears to have no strict relation with the level of the blood urea. When nausea and oliguria are present, the patient may have difficulty in tolerating the sodium benzoate and in passing urine when required. Where renal function tests reveal failure to excrete nitrogenous products, the hippuric acid excretion is obviously no guide to the condition of the liver.

Cardiac Failure.

The results in the cases of cardiac failure are shown in Table V. The symptoms and signs of hepatic involvement are so extremely common in congestive cardiac failure that it is not surprising to find low values for the hippuric acid excretion. In some, renal damage probably plays a part (Cases V, VI and VII). When jaundice was present (Cases II, VI, VII and VIII) the excretion was always much diminished; but equally low values were found in some cases not associated with jaundice (Cases X and XIII). Five patients with neither ascites nor liver enlargement (Cases I, V, XI, XII and XIV) all excreted two or more grammes of sodium benzoate. In Case XI the patient, a man, aged twenty-two years, was free from œdema at the time of the test and excreted 2·8 grammes. He died of acute pulmonary œdema three weeks later, and only œdematous change was found in his liver. This was in contrast with the four other subjects who came to autopsy (Cases VI, VIII, IX and X). All of these had long-standing congestion with permanent "nutmeg" changes in the liver, and their figures for benzoic acid excretion ranged from 0·7 to 1·8 grammes.

Toxic and Non-Toxic Goitre.

In toxic and non-toxic goitre the test was performed a few days after the patient's admission to hospital, if possible before iodine treatment was commenced, and was repeated seven to ten days after operation. Nineteen patients with diffuse toxic goitre were examined, of whom twelve excreted less than 2·7 grammes of benzoic acid before operation. After operation seven of these twelve patients excreted normal amounts of benzoic acid and the excretion of five did not return to normal. Three patients who had normal excretion before operation showed considerably lowered benzoic acid excretion after operation. Of twelve patients with nodular toxic goitre, seven showed diminished excretion before operation; five of these excreted normal amounts after operation. Of the patients with non-toxic goitre, one excreted a diminished amount of benzoic acid before operation and six patients had a normal excretion. After operation 3·0 grammes of benzoic acid were excreted by the first patient, but the amount excreted by one of the normal patients was diminished. An additional thirteen patients were examined by this test before operation, but the test could not be repeated after operation. Nine of these thirteen patients showed a diminished output of benzoic acid.

An additional nine patients with toxic goitre were examined by this test before operation, but the test could not be repeated after operation. Seven of these nine patients showed a diminished output of benzoic acid.

No correlation could be found between the result of the test and the duration of the disease, the patient's loss of weight or the basal metabolic rate.

TABLE IV.

Renal Impairment.

Case.	Patient's Sex and Age. (Years.)	Diagnosis.	Milligrammes of Urea per 100 Cubic Centimetres of Blood.	Maximum Urea Concentration. (Percentage.)	Takata-Ara Reaction.	Grammes of Benzoic Acid Excreted in Four Hours.
I	M. 19	Subacute nephritis.	75	1·5		2·0
II	M. 57	Subacute nephritis.	58			1·9
III	F. 27	Subacute nephritis.	114	1·2	No reaction.	2·2
IV	F. 40	Subacute nephritis.	27	2·0	Moderate.	1·5
V	M. 35	Acute nephritis (mild).	43	1·2	Weak.	2·5
VI	F. 51	Chronic nephritis.	77	2·8		2·6
VII	F. 50	Chronic nephritis.	74	1·25	Strong.	1·1
VIII	F. 24	Ascites; ? nephritis.	20	1·0	Weak.	2·2
IX	F. 19	Plumbism; nephritis.	39	1·0	No reaction.	0·9
X	M. 51	Gout; nephritis.	139			1·0
XI	M. 57	Carcinoma of kidney; metastasis in liver and lung.	52	0·65		1·6

TABLE V.
Heart Disease.

Case No.	Patient's Sex and Age. (Years.)	Diagnosis.	Ascites.	Liver Enlargement.	Van den Bergh Reaction.		Takata-Ara Reaction.	Grammes of Benzoic Acid Excreted in Four Hours.	Clinical Outcome.	Operative or Autopsy Findings.
					Qualitative.	Quantitative (Units.)				
I	F. 63	Hypertension.	–	–	No reaction.	—	No reaction.	2·0		
II	F. 55	Congestive cardiac failure.	+	–	Biphasic.	7·0	Strong.	0·5	Death five days later.	
III	F. 40	Auricular fibrillation; congestive failure; *diabetes mellitus*.	+	+				0·4		
IV	M. 30	Rheumatic carditis; congestive failure.	+	+	Delayed.			1·1		
V	M. 70	Coronary sclerosis; congestion, not severe.	–	–				2·5		
VI	M. 52	Coronary occlusion; congestive failure.	–	+	Delayed.	4·5		0·7	Death.	Cardiac infarction and failure; liver congested, nutmeg appearance.
VII	M. 58	Hypertension; cardio-renal failure.	–	+	Biphasic.	4·0		<1·4 1·6	Condition improved.	
VIII	M. 52	Syphilitic aortitis; congestive failure.	+	+	Delayed.	5·0	No reaction.	1·7	Death.	Cardio-vascular syphilis. Liver large, firm, with linear streaking and nutmeg changes.
IX	F. 40	Rheumatic carditis; congestive failure.	–	+	Delayed.	3·0	No reaction.	1·8	Death five months later.	Liver congested with fatty degeneration, nutmeg appearance.
X	F. 54	Atherosclerosis; congestive failure.	–	+	Delayed.		No reaction.	1·0	Death.	Atherosclerosis. Liver very large and friable, nutmeg change and fatty infiltration.
XI	M. 22	Rheumatic carditis; no congestion at time of test.	–	–	Delayed.	3·0	No reaction.	2·8	Death in acute failure three weeks later.	Liver large, pale, œdematous, firm.
XII	M. 40	Chronic alcoholism; cardiac failure.	–	+	No reaction.		Strong.	2·4	Improved.	
		After recovery nine months later.	–	–	No reaction.		No reaction.	3·0		
XIII	F. 63	Auricular fibrillation; congestive failure.	+	–			No reaction.	0·9		
XIV	F. 55	Hypertension; congestive failure; *diabetes mellitus*.	–	–			Strong.	2·4		

Miscellaneous Diseases.

A variety of pathological conditions is represented in the remainder of the cases in Table VI. Four patients with hæmolytic jaundice and two with hypoplastic anæmia showed no impairment of the hippuric acid excretion. Three patients (Cases VII, VIII and IX) with pernicious anæmia gave somewhat low figures and two patients (Cases X and XI) with anæmia associated with gastro-intestinal disease and hepatomegaly showed poor excretion. De Lor and Reinhart[3] also found diminished output in certain anæmias. Of three patients with Hodgkin's disease, one showed a diminished excretion. It is apparent from Cases I to XVI that a considerable degree of anæmia is compatible with a normal conjugating power of the liver (for example, Cases V and XVI). The fact that the liver normally works at the diminished oxygen tension of the portal venous blood may be relevant to this.

The physician is often faced with a diagnostic problem when a patient has had hæmatemesis and the opaque meal examination fails to reveal an ulcer. In three cases of peptic ulcer and four cases of hæmatemesis of obscure origin (Cases XVII to XXIII) the benzoic acid excretion lay within a normal range. These may be compared with Cases XXVIII, XXXV and XLI of Table II, in which the hæmatemesis was associated with cirrhosis, and in which values of 2·0, 2·2 and 2·4 grammes were obtained for the benzoic acid excretion. One patient suffering from *tabes dorsalis* showed a slight impairment of excretion (Case XXV). Of four patients with alcoholism unassociated with clinical liver damage, one only showed a serious impairment of excretion, and that was a woman with Korsakow's psychosis (Case XXVII). Moderately impaired excretion was found in isolated cases of pulmonary tuberculosis, rheumatoid arthritis (Case XXXV), iridocyclitis with large liver and spleen (Case XXXIX) and mental deficiency with enlarged liver (Case XLIII). A normal value was obtained in a case of arsenical dermatitis unassociated with clinical evidence of liver damage. Of seven patients suffering from *diabetes mellitus*, one showed a serious impairment of excretion persisting after treatment; the others gave normal results to the test. The hippuric acid excretion before and after stabilization and its relation to diet and insulin resistance might repay study.

Discussion.

In the examination of any test, the main factors to be assessed are its reliability, its sensitivity, its ease and safety of clinical application and its aid in the diagnosis, treatment and prognosis in an individual case.

Although diminished function does not always go hand in hand with structural change, in no case in this series was there any grave discrepancy between the results of the hippuric acid excretion test and the operative or autopsy findings. Two cases may be cited as examples. In one case of ascites a tentative diagnosis of cirrhosis was made, but the amount of benzoic acid excreted was greater than expected (2·4 grammes); at autopsy the ascites was found to be due to thrombosis of the inferior *vena cava*, the liver presenting only congestion and friability. In the second case, a woman suffering from adhesive mediastinopericarditis presented an unusually low result to the test (0·8 gramme); the autopsy revealed miliary tuberculosis with consequent toxic spoiling.

It should be remembered that the test is not reliable when the kidneys fail to excrete nitrogenous metabolites; but this difficulty seldom arises in practice. In doubtful cases the estimation of the blood urea content and of the urea clearance or concentration would detect errors due to this cause. Besides the lowered function of hepatic disease and congestive heart failure, the test revealed impaired function in certain conditions in which toxic changes are widespread—for example, pulmonary tuberculosis, pernicious anæmia and rheumatoid arthritis.

TABLE VI.

Miscellaneous Conditions.

Case Number.	Patient's Sex and Age. (Years.)	Diagnosis.	Van den Bergh Reaction.		Other Data.	Takata-Ara Reaction.	Grammes of Benzoic Acid Excreted in Four Hours.
			Qualitative.	Quantitative. (Units.)			
I	F. 51	Familial acholuric jaundice.	Biphasic.	5·5	Hæmoglobin 66%.	Weak.	3·2
II	M. 18	Familial acholuric jaundice.	Delayed.	5·0	Hæmoglobin 106%.		3·7
III	M. 14	Familial acholuric jaundice.	Delayed.	4·0	Hæmoglobin 66%.	Weak.	3·3
IV	M. 38	Chronic acquired hæmolytic jaundice.	Delayed.	3·0	Hæmoglobin 117%.	No reaction.	4·1
V	M. 52	Hypoplastic anæmia; hepatomegaly.			Hæmoglobin 40%.		3·1
VI	F. 40	Hypoplastic anæmia.					3·6
VII	F. 74	Pernicious anæmia.	Delayed.	4·0	Hæmoglobin 44%.	Weak.	1·7
VIII	F. 49	Pernicious anæmia.					2·6
IX	M. 63	Pernicious anæmia.	Delayed.	4·0	Hæmoglobin 62%.	Weak.	2·7
X	F. 46	Anæmia following gastro-enterostomy; hepatomegaly.	Delayed.	2·5	Hæmoglobin 51%.		1·4
XI	F. 47	Ulcerative colitis; hepatomegaly.			Hæmoglobin 49%.		1·0
XII	M. 40	Hodgkin's disease; jaundice.			Hæmoglobin 103%.		2·9
XIII	M. 65	Hodgkin's disease; jaundice; chronic pulmonary tuberculosis.	Delayed.	3·0	Hæmoglobin 67%.		2·3
XIV	F. 33	Hodgkin's disease.	Delayed.	2·0		Moderate.	3·2
XV	M. 36	Myeloid leuchæmia.			Hæmoglobin 62%.		4·0
XVI	M. 64	Lymphosarcoma; hæmolytic anæmia.	Delayed.	3·0	Hæmoglobin 43%.		3·0
XVII	M. 40	Peptic ulcer; hæmatemesis.					3·4
XVIII	M. 37	Hæmatemesis.					3·1
XIX	M. 55	Peptic ulcer; hæmatemesis.			Hæmoglobin 65%.		2·9
XX	M. 47	Hæmatemesis; ? acute peptic ulcer.			Hæmoglobin 90%.		3·6
XXI	M. 49	Hæmatemesis; ? cause alcoholism.				No reaction	3·2
XXII	F. 59	Hæmatemesis; ? cause.			Hæmoglobin 55%.		3·1
XXIII	M. 60	Peptic ulcer.				No reaction.	2·8
XXIV	M. 26	Œsophageal varices.			Hæmoglobin 99%.		3·7
XXV	M. 57	*Tabes dorsalis.*			Wassermann reaction +++.		3·1
XXVI	F. 37	*Tabes dorsalis.*			Wassermann reaction +++	No reaction.	2·6
XXVII	F. 44	Alcoholic peripheral neuritis.				14/3/41: weak.	3·2
						30/6/41: moderate.	2·9
XXVIII	F. 43	Alcoholic peripheral neuritis; Korsakow's psychosis.				Weak.	1·0
XXIX	M. 47	Convalescent from bronchopneumonia; alcoholism.					3·9
XXX	M.	Alcoholic gastritis.					3·4
XXXI	M. 57	*Carcinoma coli.*					2·7
XXXII	M. 50	Carcinoma of kidney; thrombosis of inferior *vena cava.*	Biphasic.	6·0	Ascites.	Moderate.	2·4
XXXIII	F. 52	Diaphragmatic pleurisy with effusion.	Delayed.	3·0		Weak.	3·0
XXXIV	F. 30	Miliary tuberculosis.	Biphasic.	5·0		Strong.	0·8
			Biphasic.	5·0			0·7
XXXV	F. 30	Bilateral pulmonary tuberculosis.				Strong.	2·4
XXXVI	F. 50	Rheumatoid arthritis.				Moderate.	2·3
XXXVII	F. 61	Rheumatoid arthritis.					2·8
XXXVIII	M. 26	Chronic appendicitis.	No reaction.				3·9
XXXIX	F. 57	Irido-cyclitis.	No reaction.		Liver and spleen enlarged.		2·0
XL	M. 24	Benign tertian malaria.	Delayed.	2·5			2·8
XLI	M. 65	Post-operative asthenia.[1]			Hæmoglobin 66%.		2·6
XLII	M. 60	Arsenical dermatitis.					3·2
XLIII	M. 31	Mental deficiency; macrocytic anæmia.	Delayed.	4·0	Liver enlarged.	No reaction.	2·4
XLIV	M. 35	Cardiac neurosis.				No reaction.	3·3
XLV	F. 53	*Diabetes mellitus*—					
		(*a*) Before stabilization.				(*a*) No reaction.	1·4
		(*b*) After stabilization.				(*b*) No reaction.	3·1
XLVI	M. 43	*Diabetes mellitus* (stable).				Weak.	3·0
XLVII	M. 39	*Diabetes mellitus* (stable).				Weak.	3·2
XLVIII	M. 56	*Diabetes mellitus* (stable).				No reaction.	1·0
XLIX	M. 69	*Diabetes mellitus* (unstable).				No reaction.	2·7
L	F. 69	*Diabetes mellitus* (unstable).				Weak.	3·0
LI	M. 65	*Diabetes mellitus.*					3·0

[1] Intermittent icterus.

Besides the judgement of the autopsy room, comparison of the hippuric acid test with other tests of liver function is a useful check on its reliability. De Lor and Reinhart[9] found the hippuric acid and bromsulphthalein excretion and the plasma prothrombin content to be the most reliable and Cates[10] the bromsulphthalein excretion, plasma protein content and hippuric acid excretion. Wilson[11] found a close correlation between the level of the plasma prothrombin and the hippuric acid excretion in 41 cases of liver disease without obstruction to the ducts. This correlation did not hold for the bromsulphthalein excretion and galactose tolerance tests.

The charge is frequently made that since the functional reserve of the liver is so great, a test will reveal diminished function only in the presence of considerable liver damage. But the hippuric acid test measures the maximum power of the liver to convert benzoic to hippuric acid; the liver is called upon to make an effort well beyond the normal physiological requirements. In this way the test is comparable to the urea clearance and concentration tests. It is probable that a liver which shows a moderate impairment in dealing with a large dose of sodium benzoate may be quite adequate for the average demands of metabolism. Even quite pronounced impairment seems compatible with months or years of life. The impairment is none the less a sign of diminished capacity to function and by inference of liver damage.

Another objection raised to the test is that the formation of hippuric acid tests only one of many functions of the liver; but the work of authors quoted above seems to show that a diminished conjugating power of the liver is associated also with defective prothrombin production, dye excretion and carbohydrate metabolism. The apparent lack of localization of function in the liver would lead one to expect this.

The test is clinically no more difficult to perform than the urea concentration test. Reasonable care is required in seeing that the full dose of sodium benzoate is taken and in the collection of specimens. Nausea occasionally occurs, but is much diminished if the patient has a light

breakfast first and is kept in bed for the first two or three hours. With these precautions it is rare for vomiting to interfere with the performance of the test. It appears absolutely harmless and in this has an advantage over the bromsulphthalein excretion test.

The hippuric acid excretion test sets out to be purely a test of function, and any diagnostic aid it gives must be only incidental. Thus ascites or liver enlargement may be due to malignant disease, cardiac failure or cirrhosis, in any of which the excretion will probably be low. Jaundice due to toxic hepatitis or the late stages of cirrhosis is associated with a poor excretion (an amount usually less than 1·5 grammes). Long-standing obstruction may produce an equally poor function; but recent obstruction is usually associated with the excretion of over 2·5 grammes. Where the test may prove useful is in the investigation of hæmatemesis when the opaque meal examination fails to reveal an ulcer. A lowered hippuric acid excretion in such a case would favour the diagnosis of cirrhosis.

It is as an aid to treatment and prognosis that the test should find its widest application. Particularly the tests performed in cases of toxic hepatitis show that it is a sensitive indicator of improvement in liver function—more sensitive than the degree of jaundice. It provides a method of comparing the effectiveness of different lines of treatment. It has a wide sphere of use in the preparation for operation of patients with jaundice. Through it Boyce and McFetridge[9] have produced confirmatory and conclusive evidence of the value of glucose in the treatment of acute liver damage. As an aid to prognosis the test appears most valuable when estimations are made at regular intervals. For patients with cirrhosis, a proper correlation of the amount of hippuric acid excreted with the expectation of life will require years of observation; but it appears certain that an excretion of amounts persistently below one gramme is associated with a bad immediate prognosis.

Fifty patients with toxic or non-toxic goitre were examined, of whom 29 showed diminished excretion of benzoic acid before operation. The results obtained after operation, while frequently indicating an improvement in the amount of benzoic acid excreted, also revealed some sharp falls in excretion.

The test has further important potentialities for the investigation of liver involvement in various diseases. This has already been begun for thyreotoxicosis; other conditions which suggest themselves are the toxæmias of pregnancy, *diabetes mellitus*, the anæmias and rheumatoid arthritis.

Takata-Ara Test.

The Takata-Ara test has had a curious history. It was introduced in 1925 by Takata[13] as a serum test to differentiate between bronchopneumonia and lobar pneumonia, and in the same year Ara[12] modified it for use with cerebro-spinal fluid. In 1929 Jezler[14] first proposed its application as a liver function test and found that it produced positive results with the serum of seven out of twelve and with the ascitic fluid of eight out of nine patients suffering from cirrhosis of the liver. The graduated modification of the test introduced by Mancke and Sommer[15] has been the one employed in this series. The manifold variations in technique used by different clinics have made it difficult to establish the limits of normality and almost impossible to compare results. There is even confusion as to whether it should be regarded as a test of liver function or as a specific diagnostic test for cirrhosis of the liver and parenchymatous liver damage.

The Takata-Ara reaction of serum depends on the degree of precipitation obtained on the addition of varying concentrations of mercuric chloride, precipitation being increased in some conditions. The mechanism is still obscure; the most popular theory, that the flocculation is due to an alteration in the albumin-globulin ratio (Hugonot, Sohier and Marchal,[16] Schreuder,[17]) is quite unproved and is not borne out by the plasma protein estimations in several of our cases. However, in this paper no attempt has been made to establish the mechanism of the test.

According to the technique of Mancke and Sommer the serum and reagents are set out in eight test tubes as shown in Table VII. After the addition of each reagent, the tubes must be thoroughly shaken. The tubes are allowed to stand overnight and the result is then read. The tubes may present heavy or moderate precipitation, heavy or moderate opalescence, or a clear solution. For convenience in reading these may be designated as grades 4, 3, 2, 1 and 0 respectively.

Normal Subjects.

Mancke and Sommer[15] state that with normal serum none of the tubes should show flocculation, but they give no account of how that conclusion was reached. We performed the test with the serum of fifty healthy persons and found precipitation in the first tube in 60% to 70% and in the first two tubes in 10%, while slight precipitation in the third tube was not uncommon. We therefore regarded heavy precipitation occurring in the first three tubes as a weak positive reaction and occurring in five or more tubes as a strong positive reaction. A weak positive reaction was obtained in such a variety of conditions as to show that it is of no significance for the diagnosis of liver disease.

Diseases of the Liver and Gall-Bladder.

In Table VIII are set out the results of tests performed on 103 patients, of whom 46 were suffering from conditions affecting the liver or bile ducts. Of five cases of jaundice due to toxic hepatitis, catarrhal jaundice and secondary syphilis, a strong or moderate reaction was obtained in four. The serum of the patient with syphilitic hepatitis returned to normal after treatment. In ten cases with cirrhosis of the liver, a strong or moderate reaction was obtained in eight and no reaction in two. The serum of four patients with the diagnosis of hæmochromatosis gave variable results. Two patients with carcinoma of the bile ducts both came to autopsy. The subject whose serum gave a strongly positive Takata-Ara reaction was found to have coexisting cirrhosis; in the case of the other no reaction was obtained and cirrhosis was absent.

TABLE VII.

Content of Test Tubes.	Number of Test Tube.							
	1	2	3	4	5	6		8
Serum (cubic centimetres)	0·1	0·1	0·1	0·1	0·1	0·1	0·1	0·1
0·9% Sodium chloride solution (cubic centimetres)	1·0	1·1	1·2	1·3	1·4	1·5	1·6	1·7
10% Sodium carbonate solution (cubic centimetre)	0·4	0·4	0·4	0·4	0·4	0·4	0·4	0·4
0·25% Mercuric chloride solution (cubic centimetre)	1·0	0·9	0·8	0·7	0·6	0·5	0·4	0·3
Resulting concentration of mercuric chloride in milligrammes *per centum*	100	90	80	70	60	50	40	30

Of the fourteen cases of cholelithiasis, both with and without jaundice, the test produced no reaction in twelve, and in the remaining cases weak positive reactions were obtained. Moderate reactions were noticed in one case of stricture of the common bile duct and in one case of carcinoma of the pancreas, and a strong reaction in one out of four cases of secondary carcinoma of the liver. In three cases of hydatid cyst of the liver the test produced no reaction and in one a strongly positive reaction.

Extrahepatic Disease.

Among 57 patients with pathological conditions not arising from the liver strongly positive reactions were obtained with the serum of eight and moderate reactions from that of seven. The cases were not a random selection, so that the figure of 25% of significant reactions among extrahepatic conditions is undoubtedly too high. The most important group is that of twelve patients with cardiac failure; the serum of four of these gave a strongly positive reaction and of two a moderately positive reaction. The reaction was not confined to the patients with jaundice. Strong or moderate reactions were also obtained in three out of ten cases of renal disease, in two out of five cases of tuberculosis and in single cases of Hodgkin's disease, carcinoma of the kidney, rheumatoid arthritis and typhus fever.

TABLE VIII.
Takata-ara Reaction.

Pathological Condition.	Number of Cases.	Strong Positive Reaction.	Moderate Positive Reaction.	Weak Positive Reaction.	No Reaction.
Catarrhal jaundice	2	1	1	—	—
Toxic hepatitis	2	1	—	—	1
Syphilitic hepatitis—					
(a) Before treatment	1	1	—	—	—
(b) After treatment	1	—	—	—	1
Cirrhosis of liver	10	4	4	—	2
Cirrhosis and carcinoma of bile ducts	1	1	—	—	—
Hæmochromatosis	4	1	1	1	1
Cholelithiasis—					
(a) Without jaundice	4	—	—	—	4
(b) With jaundice	10	—	—	2	8
Stricture of common bile duct	2	—	1	—	1
Carcinoma of pancreas	1	—	1	—	—
Carcinoma of bile ducts	1	—	—	—	1
Secondary carcinoma of liver	4	1	—	2	1
Hydatid disease	4	1	—	—	3
Cardiac failure—					
(a) Without jaundice	4	2	—	—	2
(b) With jaundice	8	2	2	—	4
Renal disease	10	1	2	2	5
Diabetes mellitus	8	—	—	4	4
Pulmonary tuberculosis	4	1	—	1	2
Miliary tuberculosis	1	1	—	—	—
Chronic alcoholism	4	—	—	2	2
Hæmolytic jaundice	4	—	—	2	2
Pernicious anæmia	3	—	—	2	1
Hodgkin's disease	2	—	1	—	1
Carcinoma of kidney	1	—	1	—	—
Carcinoma of stomach	1	—	—	—	1
Peptic ulcer	1	—	—	—	1
Chronic appendicitis	1	—	—	—	1
Pneumococcal meningitis	1	—	—	1	—
Rheumatoid arthritis	1	—	1	—	—
Tabes dorsalis	1	—	—	—	1
Anxiety neurosis	1	—	—	—	1
Typhus fever	1	1	—	—	—

Toxic and Non-Toxic Goitre.

The Takata-Ara test was performed on the serum of forty patients before operation; two specimens gave a moderately positive reaction, the remainder giving weak reactions or no reactions. The two patients whose serum reacted to the test gave normal results to the hippuric acid tests. No significant change occurred after operation in the reaction of the serum of the forty patients.

Discussion.

The value of the Takata-Ara test must be assessed from two standpoints: First, is it of value as a test of liver function, as claimed by Jezler?[18] Second, what is its diagnostic and prognostic import?

The first point we have attempted to answer by a comparison of the Takata-Ara and hippuric acid tests and the clinical progress of the patients. From Table II it can be seen that 29 Takata-Ara tests were performed in these cases of liver disease: in eight the reactions were strongly positive and the amount of benzoic acid excreted ranged from 0·8 gramme to 3·3 grammes (average, 1·6 grammes); in seven the reactions were moderately positive and the amount of benzoic acid excreted ranged from 0·8 gramme to 3·6 grammes (average, 2·0 grammes); and in fourteen the reactions were weakly positive or absent and the amount of benzoic acid excreted ranged from 0·6 gramme to 3·2 grammes (average, 2·2 grammes). The discrepancy between the results of this test and the liver function is even more apparent in the series of tests on patients with disease of the gall-bladder and ducts (Table III), in which only one out of fourteen tests produced a significant reaction, even in the presence of considerably diminished liver function.

That the Takata-Ara test is not specific for cirrhosis of the liver or parenchymatous liver disease has been acknowledged by nearly all workers (Bowman and Bray,[20] Chasnoff and Solomon,[20] Hugonot, Sohier and Marchal[18]). It is sufficiently apparent from this series. The percentage of positive reactions among cases of cirrhosis must depend on the severity of the disease and the method employed. Ragis[21] found the reaction positive in 98% of cases, and Chasnoff and Solomon[20] hold that an absence of reaction would throw doubt on the diagnosis of cirrhosis. On the other hand, Magath,[22] in a recent review, points out that the reaction is frequently not obtained in early cases (as in two of our series) when diagnostic aid is most required. Sommer[23] suggested that the test might be useful in differentiating parenchymatous and obstructive jaundice. His view receives considerable support from this series, since in 15 out of 20 cases of parenchymatous disease strong or moderate reactions were obtained, whereas in only two out of fourteen cases of obstructive jaundice were moderate reactions obtained.

It appears therefore that the Takata-Ara test alone is unreliable either as a test of liver function or as diagnostic of liver disease. In conjunction with other tests (such as the Van den Bergh test and hippuric acid excretion test) a strongly positive reaction would confirm a diagnosis of parenchymatous disease of the liver. In a case of jaundice a strongly or moderately positive Takata-Ara reaction is in favour of a non-obstructive origin for the jaundice. An absence of reaction is of little significance.

Summary.

A series of cases is reported of 313 hippuric acid excretion tests performed on 240 adult subjects. An attempt is made to assess the value of the test and its clinical application.

The serum of 143 patients and of 50 normal subjects has been tested by the Takata-Ara reaction. The findings are discussed.

Conclusions.

1. Quick's hippuric acid excretion test is both a reliable and a sensitive index of liver function, and is a valuable aid in estimating clinical progress, response to treatment and prognosis.
2. It is simple to perform and devoid of danger to the patient, but is not applicable to patients with poor renal function.
3. The Takata-Ara test is practically valueless as a test of liver function and is not a specific test for liver disease. It is, however, a useful confirmatory test for parenchymatous liver disease.

Acknowledgements.

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AN INVESTIGATION OF HEPATIC FUNCTION IN THYREOTOXICOSIS.

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DURING the last few years tests for certain of the liver functions have undergone modifications and refinements and now give a more accurate measure of these hepatic functions. One may instance the galactose tolerance test for the glycogenic function which has undergone many alterations. Lichtman (1932) compared the urinary excretion of galactose of normal persons with that of thyreotoxic patients and found that thyreotoxic patients had a normal rate. On the other hand, Althausen and Weaver, using a technique whereby the galactose in the blood was estimated, were the first to find evidence of impaired function in thyreotoxic states. Maclagen (1940) introduced further refinements, and together with Rundle, Mills and Collard (1940), reported results which were interpreted as striking evidence of liver damage in both clinical and experimental thyreotoxicosis.

Another instance is that of the hippuric acid excretion test developed by Quick (1933) following on Bryan's studies in 1925.

Boyce and McFetridge (1938), Lichtman (1938) and Bartels (1938) have reported positive results. However, since these investigations were carried out the hippuric acid test has been considerably refined by the work of Weichselbaum and Probstein (1939).

The object of this paper is to report investigation of the detoxicating function of the liver of patients suffering from thyreotoxicosis by means of the new, more accurate technique, and to compare and contrast the results with those obtained by the blood galactose tolerance test.

THE LIVER IN THYREOTOXICOSIS.

Much evidence is available in the literature to show that the liver is damaged in many cases of thyreotoxicosis, sometimes to a severe degree. Jaundice has been recognized for many years as a complication of exophthalmic goitre, and Habershon (1874) reported the first fatal case. In 1898 Sutcliff observed jaundice in a severe case of exophthalmic goitre, and Eder in 1906 reported three cases. According to Beaver and Pemberton, Assmann (1931) stated that "signs of hepatic disturbance, especially icterus, may occur in cases of exophthalmic goitre, and icterus may be present without having a causal relationship to the exophthalmic goitre as a manifestation of catarrhal jaundice, as a result of cholelithiasis or due to inflammatory conditions of the biliary tract. The icterus, however, may be an immediate complication of the exophthalmic goitre when there is cardiac decompensation, or it may occur independently of a failing heart as the result of a direct toxic influence of the hyperthyroidism." Assmann concludes that icterus as a complicating symptom of exophthalmic goitre is found mainly in the most serious cases. The cases, however, do not necessarily terminate fatally. Many other reports of jaundice associated with toxic goitre are to be found in the literature. However, too much importance must not be laid upon it, as the condition is rare and was found in two cases only out of a series of 450 at the Royal Prince Alfred Hospital, Sydney (Wyndham, 1940).

Autopsy studies on subjects dying of thyreotoxicosis have revealed pronounced liver damage in most cases, and even acute yellow atrophy has been recorded. Kerr and Rusk (1922) described a case in which jaundice developed and at the post-mortem examination a condition of acute yellow atrophy of the liver was found. Kerr (1930) stated that he had observed several other cases in addition to the one previously reported. Raab and Terplan (1923), Barker (1930), Assmann (1931) and Wyndham (1940, two cases) all report cases of acute yellow atrophy. Lesions of a lesser degree are exceedingly common and have been studied particularly by Beaver and Pemberton and by Weller. A recent report has been given by Wyndham (1940).

Beaver and Pemberton have reported on a series of autopsies in 107 cases of exophthalmic goitre. They find that three types of lesions predominate in the liver: (i) Acute lesions, comprising fatty metamorphosis, focal and central necrosis, and changes secondary to blood stasis; these occur in varying degrees in over 90% of cases. (ii) Simple atrophy, which occurs in a significant number of cases (63%). (iii) Subacute atrophy and cirrhosis, occurring in approximately 60%. This last lesion is similar to that described by Weller (1933) as "patchy chronic parenchymatous interlobular hepatitis"; in his cases it had an incidence of 54% against a matched control series of 20%. This lesion is of greatly varying degrees of severity, and is occasionally of an advanced type,

resembling an established cirrhosis from other causes; it must be a factor in the prognosis of these cases.

According to Beaver and Pemberton, the common factor in the production of these lesions appears to be the severity of the syndrome of exophthalmic goitre.

The acute lesions are not usually influenced in their occurrence or severity by the age of the patient, the sex or percentage loss of body weight. Except for loss of weight the lesions usually occur directly in proportion to the severity of the disease. They appear most prominently in the more severe cases of shorter than average duration.

Atrophy seems proportional to the severity of the disease, whilst duration does not seem to be a factor. The changes of subacute atrophy and cirrhosis predominate in older patients, among whom the disease is of longer duration and of more than average severity. Atrophy, acute changes and cirrhosis are probably only stages in the same pathogenic process, which has its origin in the toxic effect of the hyperthyreoid state. Wyndham obtained somewhat similar results in his investigation, but concludes that autopsy material does not suggest that the liver plays an obvious part in the deaths from hyperthyreoidism.

In addition, Moon (1936) has shown that in United States of America those States with the highest incidence of thyreoid disease also have the highest incidence of hepatic cirrhosis, and that as far as Europe is concerned, the same condition of affairs exists in Switzerland.

Experimental Studies of the Liver in Thyreotoxic States.

Experimental studies are also suggestive. Farrant (1913), feeding thyreoid gland to various laboratory animals, produced livers showing fatty degeneration. Hashimoto (1921) observed varying degrees of fatty degeneration and necrosis in rats made toxæmic with thyreoid. Maclagen, Collard, Mills and Rundle (1940) produced histological evidence of degeneration in the livers of rabbits treated with thyroxine, which led to acute and subacute necrotic changes focal in type.

On the functional aspect, Cramer and Krause (1913) showed that the liver glycogen content was greatly diminished by the administration of thyreoid gland to cats and rats. The reason that the liver is so much damaged is probably that it plays an important role in the detoxication and excretion of the thyreoid hormone.

Material and Method.

The method of Weichselbaum and Probstein (1940) was used throughout this investigation. It differs from Quick's technique in certain important features, and shows an important improvement in accuracy of the results. In actual practice it raises the normal average of benzoic acid obtained from the urine to 3·7 grammes (3·0 grammes by Quick's method). The main difference is that in the new method the residue of hippuric acid remaining in the urine is reduced to a minimum because it is rendered less soluble by the addition of large quantities of common salt. The variation in solubility of different specimens of urine is also minimized by this means. In addition, in this method a smaller dose is used, namely, 4·0 grammes of sodium benzoate given by mouth; this produces more uniform results. At first the test was used over four hours, but the output of the fourth hour is practically negligible, and the test is now carried out over three hours only.

The actual technique is as follows.

One hour after a small breakfast consisting of toast and tea (no meat should be given), the bladder is emptied and 4·0 grammes of sodium benzoate are given by mouth in two ounces of water flavoured with peppermint and followed by a small glass of water. The total amount of urine is collected over the next three hours and pooled. Occasionally hourly specimens were examined separately. During this period nothing is taken by mouth.

The urine is tested within twelve hours, because hydrolysis will occur unless preservatives are added. The urine is measured and made up to 30% sodium chloride solution by the addition of the requisite amount of pure common salt. Heat is necessary to hasten the solution of the salt. The urine is then cooled in a cold water bath and a few cubic centimetres of 10/N sulphuric acid is added. It is then stirred vigorously with a glass rod until precipitation of the hippuric acid occurs. The urine is then allowed to stand for a further ten minutes in a cold water bath. After this it is filtered through a Hirsch funnel, suction being used, and the precipitate of hippuric acid is washed with a small amount of iced 30% sodium chloride solution to remove any excess sulphuric acid. The precipitate is then dissolved on the funnel with boiling water, and the resulting filtrate is collected and titrated whilst still hot with N/2 sodium hydroxide solution, phenolphthalein being used as an indicator.

Calculation.

The number of cubic centimetres of 0·5N sodium hydroxide solution used is multiplied by 0·072, which gives the amount of sodium benzoate in grammes from which the hippuric acid was derived. To this must be added the correction for solubility of hippuric acid, which is equivalent to 0·1 gramme of sodium benzoate per 100 cubic centimetres of urine. For instance, if the volume of urine is 200 cubic centimetres and it takes 10 cubic centimetres of N/2 sodium hydroxide solution to neutralize it, the amount of hippuric acid present (expressed as sodium benzoate) is:

$$10 \times 0{\cdot}72 + 2 \times 0{\cdot}1 \text{ gramme} = 0{\cdot}92 \text{ gramme}.$$

The average excretion of 30 normal subjects in three hours was found to be 82% of the original 4·0 gramme dose, or 3·28 grammes with a possible variation of ±8%. This was taken as a basis of comparison, and the figure 3·28 grammes was taken to represent a detoxicating liver function of 100%. It has been found more convenient to express the results as a percentage of the normal 100% rather than in grammes as has been customary in Quick's test.

Comment.

There are several fallacies with the method. In the first place, the patient may vomit the sodium benzoate, although this appears to be less frequent with the 4·0 gramme dose. Also severe degrees of renal failure interfere with the accuracy of the method as a test of liver damage, since there is a hold-up in the excretion of the hippuric acid by the damaged kidneys. Gastric retention or states of dehydration—for example, vomiting, diarrhœa, low fluid intake *et cetera*—would tend to nullify the results. It should be easy to recognize and correct any of these factors which may tend to cause fallacious results.

This test of liver function depends on the conjugation in the liver of glycine and benzoic acid to form hippuric acid. The liver plays a double role in this reaction, both manufacturing the glycine and effecting its conjugation with the benzoic acid. This is a similar reaction to the detoxication of certain toxins absorbed from the intestine, and the test is to be regarded as a measure of the detoxicating function of the liver.

The clinical material consisted of 73 cases of thyreotoxicosis of both the primary and secondary type, and 20 cases of non-toxic goitre and anxiety states. Dr. Hugh Poate, Director of Post-Graduate Surgery, was responsible for the diagnosis, routine clinical investigation and treatment. Whenever surgical treatment was instituted, the thyreoid gland was obtained at operation and examined microscopically. The post-operative reaction was noted in all cases. In no instance was the patient jaundiced. Two of the patients had auricular fibrillation and were receiving digitalis therapy.

Results.

Figure I gives a composite picture of the results of the initial test in thirty consecutive cases of thyreotoxicosis, graded according to their severity. It is obvious that in these cases there is in general a severe depression of the detoxicating function of the liver. The variation is large,

ranging from 25% function up to 100% function in a few cases. In the majority of cases the readings are well below the limit of normal range, while in some cases a profound depression of normal function is present.

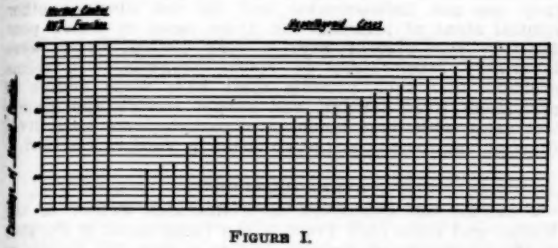

FIGURE I.

Figure II gives an idea of the excretion curves; *A* is the normal curve, *B* is the curve in a case associated with average depression of liver function, and *C* is the curve in a case associated with severe depression. It is interesting to compare these two graphs with similar graphs of the blood galactose test in the paper by Maclagan *et alii* (1940), previously mentioned. In each case the similarity of the results is striking. There are a large

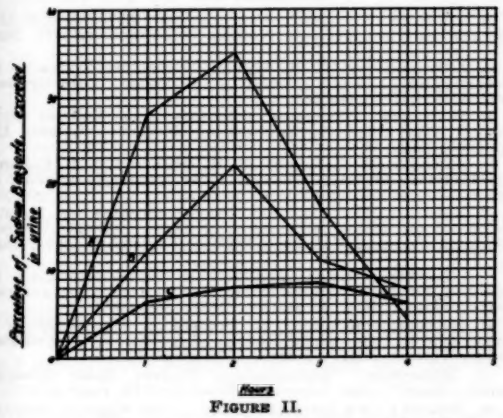

FIGURE II.

number of results in the zone of medium failure grading slowly up to a few results within normal limits. The curves in individual cases obtained by both tests also show discrepancies from the normal of similar degree.

Figure III shows that in non-toxic goitre there is a mild general decrease in liver function, falling as low as 61% in one case. As a general rule the impairment is small, and in most cases the function lies near the lower limits of normal. This shows that hippuric acid excretion cannot be used as a diagnostic test in suspected cases of thyreotoxicosis. Only five cases of anxiety neurosis were tested and in each case there was no impairment of function.

The Effect of Treatment by Rest Sedation and Iodine Therapy.

The reaction to treatment is shown in twelve cases in Table I. The patients may be divided into two groups: (i) those who show considerable improvement—this is the usual reaction; (ii) those who give consistently poor results to the test and may show little or no improvement, or even retrogression—these are uncommon.

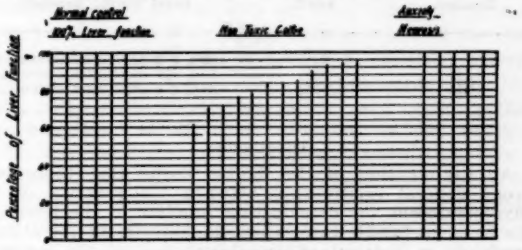

FIGURE III.

It is to be noted that in the first group the improvement seems to keep pace with other clinical signs of amelioration, such as lowering of the basal metabolic rate and the pulse rate. Those occasional patients with mild thyreotoxicosis associated with considerable depression of liver function usually respond rapidly and approach normal after one or two weeks of treatment. In more severe cases months may be needed. Case VIII is a case in point; approximately two months were required. In some cases improvement was very slow, although the patients were helped by a very high carbohydrate intake.

The second group, comprising patients showing little or no improvement, is a small but important one. In this series there were three such patients showing little improvement in multiple tests over many weeks; Cases VI and X are examples. Two of these three subjects came to autopsy, and an extreme degree of damage was found in the liver parenchyma. Other clinical signs, however, showed that the patients were "bad risks". This group will be mentioned later in the discussion of the significance of this liver function.

Post-Operative Reaction.

The degree of post-operative reaction does not appear to depend on the pre-operative level of the detoxicating function of the liver. Some patients with good function show a severe reaction and some with poor function show a mild reaction. Three patients showed a severe reaction, and the figures given in Table II show a severe depression of liver function in each case following operation. In Case II the reaction was long-lived, and depression was present after fifteen days.

TABLE I.

The Effect of Treatment by Rest, Sedation and Iodine Therapy.

Case Number.	Before Treatment.			After Treatment.		
	Liver Function. (Percentage.)	Basal Metabolic Rate. (Percentage.)	Pulse Rate per Minute.	Liver Function. (Percentage.)	Basal Metabolic Rate. (Percentage.)	Pulse Rate per Minute.
I	47	+30	108	90	+2	72
II	39	+39	112	71	+5	86
III	58	+35	130	92	+18	90
IV	48	+41	122	78	+22	96
V	40	+35	106	93	1+	80
VI	46	+60	126	44	+30	106
VII	56	+29	134	60	±0	70
VIII	58	+26	140	75	−10	80
IX	79	+23	96	89	+14	86
X	28	+32	84 (Digitalis)	46	+43	86 (Digitalis)
XI	44	+26	100	84	+11	80
XII	60	−4	106	92	−25	84

Remote Post-Operative Findings.

A limited number of tests (six only) were performed some time after operation. All indicated liver function within normal limits. According to Maclagen *et alii* (1940), the galactose index indicated mild residual liver damage under similar conditions.

TABLE II.

Liver Function During Severe Reaction.

Case Number.	Pre-operative Level.	Level During Reaction.
I	84%	30% (3 days after operation)
II	93%	40% (15 days after operation)
III	49%	38% (5 days after operation)

DISCUSSION.

An examination of the figures shows that there is a broad general relationship between the severity of the thyreotoxicosis, the detoxicating function of the liver and the basal metabolic rate, when the severity of the clinical condition is determined by observation of the patient's general condition, loss of weight and pulse rate. Moreover, when adequate pre-operative treatment with iodine is given, the clinical improvement is parallel with improvement in liver function and basal metabolic rate. Although the hippuric acid excretion test concerns a different function of the liver, it gives results comparable to those given by the galactose index, and the results obtained in this investigation confirm the opinion of other observers as to its great clinical value.

It must, however, be remarked that the relation of liver function, basal metabolic rate and severity of thyreotoxicosis does not appear to be a close one. In severe this group. To be able to diagnose the occasional cases of this series in two moderately severe cases the test revealed 100% function; they were both cases of recent primary exophthalmic goitre. On the other hand, in clinically mild cases poor liver function readings may be obtained. Older patients with low-grade chronic thyreotoxicosis filled this group, and it is noteworthy that they responded well to treatment and finally had a satisfactory pre-operative function.

In the majority of cases in which improvement occurs under treatment and normal pre-operative or post-operative readings are obtained, the initial lowered reading is probably an index of an acute pathological change that tends to return to the normal; there can be no gross permanent liver damage in this group. They probably correspond to the acute catarrhal lesions; the mild atrophies and the early cases of cirrhosis of the liver.

That small group (three in number in this series) in which low readings were obtained over a long period is even more interesting. It appears probable in this group that permanent liver damage exists. Two of these patients came to autopsy. In one case advanced atrophy of the liver cells with a fine cirrhosis was found, and in the second also advanced atrophy was present along with some chronic venous congestion and fatty degeneration. These findings represent severe liver damage, but not damage to the extent shown in some of the autopsy records. Much more investigation needs to be made before the situation in these cases is clarified, and the work must be done in conjunction with an investigation of cases of cirrhosis from other causes. It is interesting to note that the results of this test obtained so far in advanced cirrhosis due to other causes correspond closely with the results in this group. To be able to diagnose the occasional cases of severe cirrhosis complicating thyreotoxicosis would be an advance, and these results are promising, especially when correlated with the results of the blood galactose tolerance test.

From a clinical point of view it appears that the liver function is of similar value to the basal metabolic rate in indicating the severity of the disease and improvement under pre-operative treatment. It appears to be of the same value as the blood galactose tolerance test, and each has the value of providing an extra physical sign to help the clinician in assessing the operative risk. Of course, they are not indispensable and do not displace other clinical signs of toxæmia. In those cases in which poor function was present over a long period, there were invariably other signs of severe toxæmia. It seems probable that such patients must have some degree of permanent liver damage, and should receive appropriate treatment with large amounts of carbohydrate until liver function improves and the situation is further clarified.

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The Medical Journal of Australia

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SNEEZING AND DROPLET INFECTION.

SOME years ago a senior physician on the staff of one of the leading Sydney hospitals in the course of some remarks on the common cold said that an acquaintance suffering from an acute coryza had come to him in a place of public assembly and in close confidence had breathed his opinion on the uselessness of doctors because they could not cure a common cold. Our erudite physician replied to his confidant that although doctors might not know how to cure colds, they knew how to prevent them, and he therefore begged to be allowed to change his seat. This he very properly did. Many a long treatise has been written about the common cold and much research has been undertaken into its nature and its cure. It is probably true, as the physician already mentioned remarked, that a cold untreated lasts for two weeks and that when it is treated it lasts for a fortnight. In the circumstances all that we can do is to prevent an infected person from transmitting his infection to someone else.

An important research into this everyday subject has recently been carried out at the National Institute for Medical Research in Great Britain. It is reported by R. B. Bourdillon and O. M. Lidwell in *The Lancet* of September 27, 1941. They have used flash photography to study the projection of droplet particles in sneezing. Apparently American workers, headed by W. M. Jennison, have shown that in an ordinary sneeze many thousands of droplets are projected at an initial velocity which may sometimes be as high as one hundred and fifty feet per second, and also that most of the droplets come from the mouth. Bourdillon and Lidwell illustrate their report with some instructive flash photographs. These show that while in the majority of sneezes most of the droplets come from the mouth, sometimes the discharge is purely nasal and that in other instances the discharge comes from both nose and mouth. The picture of the discharge from the mouth shows at some distance from the patient's face a thick nebulous cloud that might well be a section of the "milky way" photographed on a starry night. The picture of the discharge from both nose and mouth shows that it is being blown forwards by the air stream from the mouth; it does not go downwards. The size of the droplets varied with different types of sneeze. When teeth or lips were tightly closed and expiratory pressure was high, a fine spray resulted; relatively few large drops were produced when jaws and lips were open and expiration was moderated. Bourdillon and Lidwell therefore conclude that a sneeze with an open mouth is probably less dangerous to other people than a sneeze of the other type. A picture of droplets in motion enlarged from a small photograph to two-thirds of their life size is a formidable affair and may perhaps be described as like fat globules in an emulsion reflecting the light. In a vigorous sneeze numbers of droplets were found to hit a vertical plate three feet distant from and a few feet lower than the subject's mouth. The range of danger from direct projectile infection, we are told, is not less than four feet, but will rarely exceed six feet for a person at the same height as the sneeze. A series of tests were made with a subject sneezing in a small closed room and with culture plates exposed. It was shown conclusively that a large number of bacteria-carrying particles from a sneeze were small enough to remain suspended for over fifteen minutes before falling on the culture plates. Bourdillon and Lidwell point out that such particles come within the category of what Wells has called "droplet nuclei" and are certainly capable of conveying infection under certain conditions.

This brings us to the question of what we can do to save people from the effects of sneezes. Bourdillon and Lidwell indicate several measures and place them in the order of their efficiency and *The Lancet* in an editorial makes comment on some of them. To sneeze directly into a coal or gas fire will ensure sterilization of nearly all the droplets—"bad mannered but efficient", *The Lancet* remarks. A person with a coryza cannot always be at a fire side, nor can he take a fire about with him in which to consume his droplets. Thus we come to the next which is the careful use of a large handkerchief. Bourdillon and Lidwell reproduce a picture of a man sneezing into a handkerchief and of one colony grown on a culture plate after exposure to such a sneeze. If people must sneeze, this is the kind of protective measure that they must cultivate. But here handkerchiefs must be reasonably large and be changed at fairly frequent intervals. Here also it must be remarked that the tissue (paper) handkerchiefs now in common use for colds in the head are useful in that they mop up nasal secretion and can be burned, but they are not really large enough for the smothering of a big sneeze. Bourdillon and Lidwell's next method is the use of masks. Transparent masks are not effective for long and gauze masks soon become messy and unpleasant. In this regard it may be useful to recall the work of Hirschfeld and Laube on surgical masks discussed in these pages in the issue of August 2, 1941. The last method is the use of the hand which is in the editorial language of *The Lancet* little more than a polite gesture and miserably inefficient. *The Lancet* also asks, why not go one better and abolish sneezing? If it can be done, why not? It is stated that momentary firm pressure on the nose with finger and thumb just below the bridge

will completely inhibit a developing sneeze. "This is the safeguard of the burglar and of the surgical patient with stitches in his belly wall." So even crime may have its uses—the science and art of burglary may some day be looked on as having made a contribution to public hygiene.

This work reported by Bourdillion and Lidwell gives ocular proof of what was already largely known. The subject is commonplace, but none will deny its importance. Photographs such as those reproduced in this study would make excellent health propaganda. In trams, trains and other means of public conveyance it would be fitting to display pictures of a man sneezing with a section of the "milky way" in front of his face and of another sneezing with a large handkerchief suitably clasped in front of his nose and face; and of course with legends suited to the occasion.

Current Comment.

CORONARY THROMBOSIS OR MYOCARDIAL INFARCTION.

PATHOLOGISTS performing autopsies upon persons who have died suddenly and unaccountably have often reported the absence of any coronary occlusion or myocardial infarction, although some coronary sclerosis may be obvious to naked-eye examination. Again, in hospitals, occasion has been taken to examine the hearts of certain patients who have died following a typical attack of what is generally recognized in clinical circles as coronary occlusion or coronary thrombosis. Most of these patients display a necrotic myocardium, but not all. Some who have a necrotic cardiac wall have no sign of recent arterial thrombosis or blockage in the artery designed to nourish the dead area.

H. L. Blumgart and M. J. Schlesinger have investigated these anomalies and have made some revolutionary discoveries, which should result in a careful selection of terms to describe this type of acute cardiac failure.[1] Many investigators have preceded these workers in making correlative studies of clinical features and post-mortem appearances, but none has taken into account with the same care the condition of the collateral circulation at the time of death. Unless this is accurately known, any conclusion as to the relationship of obstruction to a main coronary artery and an area of muscle softening must remain indefinite. By the employment of a technique developed by Schlesinger for the display of the whole coronary arterial system in all its intrinsic relationships, he and Blumgart have described their results in a series of no less than 325 examinations. The right and left coronary arteries were injected simultaneously with differently coloured radio-opaque masses under a pressure of 150 to 200 millimetres of mercury. The heart was then "unrolled" by a special form of dissection, a skiagram was made, and finally the arteries were dissected out separately and examined throughout their distribution.

One advantage of injection of the coronary arterial trio with coloured lead agar preparations is that it is possible to form an estimate of the size of the terminal arterioles and of any communications between the left and right coronary systems, since the injection mass will not enter a channel of less than 40 microns in diameter. Blumgart and Schlesinger have been able to show that in the young healthy heart it is possible to force watery solutions from one coronary system to the other, indicating that such communications do exist, but it is impossible to cause the lead agar preparation to pass over to the opposite coronary arterial system. It is thus obvious that no intercommunications of the order of 40 microns in diameter exist in the healthy heart. If, however, narrowing of a coronary artery occurs, the larger collateral pathways develop and supply that area of the myocardium which would otherwise suffer from diminished blood flow. This development of a satisfactory anastomotic circulation is not necessarily a constant association with old age, and Blumgart and Schlesinger report that 149 patients over the age of seventy years who had died from surgical causes were free from arteriosclerosis or other abnormality of the coronary arteries. This is an important discovery and refutes some of our current conceptions on this point.

The next category of patients investigated included those who had no clinical cardio-vascular disease and yet whose hearts showed an old healed complete occlusion in at least one coronary artery. There were 38 such instances in the series, and an additional 34 showing considerable narrowing of a main coronary artery, but no actual blockage. In those cases in which electrocardiograms had been obtained, the tracings were normal. The explanation of this condition was that in each heart investigated a large and satisfactory collateral circulation had developed, as demonstrated by the free passage of injection material as a by-pass around the occluded segment of artery. Such hearts may function satisfactorily, but their reserve is greatly depleted and any further arterial obstruction or insufficiency may not be compensated for in time to save the ventricular wall from necrosis. It was obviously the slowness of the arterial occlusion which gave such hearts their chance of survival. The normal electrocardiographic findings should also serve to teach the lesson that such evidence, when negative, is without significance.

The observations of these authors in 47 cases of *angina pectoris* are in accord with the presence of relative inadequacy of the coronary circulation, which becomes insufficient for the increased requirements of exercise, emotion and so on. Among the chief factors increasing the work and nutritional requirements of the heart are valvular disease and arterial hypertension. In each case in which *angina pectoris* was the primary cardiac symptom, Blumgart and Schlesinger found old coronary arterial occlusion or narrowings. The clinical moral of this observation is worthy of restatement. In patients previously well who suddenly begin to suffer from *angina pectoris*, or in those in whom attacks of *angina pectoris* suddenly assume a new severity, the occurrence of progressive narrowing or, more often, complete occlusion of a coronary artery must be considered. In such circumstances bed rest is strongly advisable to permit sufficient time for the development of a more adequate collateral circulation.

The authors wish to introduce a new term to describe a clinical condition which is half-way between an average attack of *angina pectoris* and a classical attack of myocardial infarction. When cardiac pain is more prolonged than usual, especially after emotion or exertion, and yet the clinical evidences of organic myocardial damage, such as fever, leucocytosis, increased sedimentation rate *et cetera*, are wanting, Blumgart and Schlesinger suggest that a state of "coronary failure" exists. They state that when "coronary failure" occurs in circumstances which have not previously provoked attacks in that subject, a diagnosis of acute coronary occlusion is justifiable. It is justifiable, however, only if the above-mentioned evidences of organic muscle damage follow in a day or two. In any case, bed rest would appear to be urgently desirable. These attacks have been called "preliminary pain" and "warning pain" by other authors.

The diagnosis of myocardial infarction should be reserved until frank clinical and cardiographic evidence of muscle damage is forthcoming. Blumgart and Schlesinger emphasize the amazingly high incidence of complete former occlusions of the main coronary arteries in cases of uncomplicated *angina pectoris*, and also, and this is important, the occurrence of fresh infarction in the absence of fresh occlusions. The coronary circulation, with all its collateral adjustments, apparently often reaches a state in which any powerful influence, even exposure

[1] *The Journal of the American Medical Association*, January 11, 1941.

to cold, demanding a sudden additional coronary flow, can precipitate an acute coronary failure, with terminal myocardial infarction, but no fresh occlusion. Another lesson learned from their investigations is that obstruction by thrombosis of one main coronary artery may produce infarction "at a distance" or in an area nourished by branches of the opposite coronary artery, but where blood reaches them through collateral channels from the opposite main vessel.

The results of these studies indicate that the terms coronary occlusion and coronary thrombosis refer to an event which may give rise to various clinical syndromes or equally to none at all. The terms should therefore be dropped from the clinical vocabulary, and the diagnosis of "myocardial infarction" made when sudden severe crushing pain and collapse, with evidence of tissue necrosis and a progressively changing electrocardiogram occur under conditions when the work of the heart is not increased, for example, during sleep or "normal" activity for that individual. Acute myocardial infarction bears no necessary relation to the formation of thrombi or occlusions: either may occur in the absence of the other. The terms "*angina pectoris*", "coronary failure" and "acute myocardial infarction" conform more accurately to conditions found than the terms "coronary occlusion" or "coronary thrombosis", which, as we have seen, do not necessarily produce any characteristic clinical manifestations. The changes in the myocardium depend solely on the extent and duration of the relative ischæmia, itself a factor of numerous mechanisms, and not on the manner in which the changes are produced.

We are indebted to Blumgart and Schlesinger for an effective and convincing demonstration of a number of important cardiological facts, long suspected on clinical grounds, but yet awaiting demonstration by their careful and incontravertible technique.

THE PERMEABILITY OF FIBROUS TISSUE TO THE TUBERCLE BACILLUS.

In tuberculosis of long standing the tubercles become encapsuled with fibrous tissue. The lesions of so-called arrested tuberculosis may by this means be apparently shut off entirely from the surrounding tissues. It has been argued that the organisms are firmly imprisoned by these fibrous walls and cannot escape unless the walls are breached by violence. An experiment devised for testing the validity of this theory has recently been conducted by Diran Yegian and Henry Halley, junior.[1] They point out that doubt has been cast on the theory because at the examination of the lungs of persons who have died of some disease other than tuberculosis, minute recent lesions may often be found close to what might be termed residual lesions, "suggesting that the recent lesions were produced by the escape of bacilli through the capsule of the older". They used sixty-one guinea-pigs in their experiment. Twenty-five of these had been sensitized by inoculation with a suspension of tubercle bacilli; the remaining thirty-six were healthy and had not been sensitized. A transverse incision about one centimetre long was made through the skin and subcutaneous tissues of the animal and by blunt dissection a channel four centimetres long was made between the subcutaneous tissue and the underlying structures. A rubber tube, four millimetres in diameter, was passed into the channel, its outer end being fixed to the skin by a suture of copper wire two centimetres from the wound. In several instances, despite the greatest care, the wounds became infected. These animals were excluded from the investigation. The tube was removed from the remaining animals after a period of six, thirty or sixty days. The wounds were clear and there was no bleeding. Immediately on the removal of the tube a saline suspension of 0·1 milligramme of virulent tubercle bacilli was allowed to flow into the channel. The mouth of the channel was plugged with cotton, and several layers of gauze were placed over the channel and fixed in position by a band of adhesive tape encircling the animal's body. Ten control animals were smeared with a heavy suspension of tubercle bacilli on the skin of the interscapular region and similarly bandaged; after a period of three months none of these animals showed evidence of tuberculosis; therefore it may be accepted that infection by way of the skin or mouth does not occur when this technique is used. The animals that had been operated on were killed from one to twelve days after the implantation of the bacilli in the channels. The lymphatic glands of axillæ and groins were removed and ground in a mortar and emulsified. Part of the emulsified tissue was injected into a healthy guinea-pig and part was stained for tubercle bacilli. The channel of fibrous tissue was removed, fixed and sectioned.

The results of subsequent examination were remarkable. In many instances tubercle bacilli were found in the fibrous wall of the channel, and in the great majority the lymphatic glands were found to be infected. After the tube had been left in position for sixty days, the wall of the channel had become quite thick and densely fibrous; yet in some instances the organisms were found in the wall. Even when the animal was killed one day after the instillation of the emulsion of tubercle bacilli into the channel, infection of the draining lymphatic glands was demonstrated. There was little difference in the results whether the animals had been sensitized or not.

Yegian and Halley express the opinion that the results of their experimental work justify the conclusion that "tubercle bacilli pass through fibrous tissue which is analagous to the fibrous tissue present in the capsule of the tubercle".

This was apparently a very carefully conducted experiment. At the same time, it should be borne in mind that the natural tubercle and the lesion produced by Yegian and Halley are by no means analagous. Medical men should keep an open mind on the question of permeability of the tubercule's capsule and should look with suspicion on every tuberculous lesion however well healed it may appear to be.

[1] *The American Review of Tuberculosis*, November, 1941.

SULPHUR AND DRUGS OF THE SULPHANILAMIDE GROUP.

There are still many medical practitioners who forbid aspirin, sulphates and food with a high sulphur content to patients taking drugs of the sulphanilamide group, although it has been shown that the abnormal pigment that sometimes appears in the blood is not usually the dangerous sulphæmoglobin. The proper use of the sulphanilamides is of sufficient importance to warrant a comment here on the results of a recent investigation made in India by W. McN. Niblock.[1] Niblock points out that Smith administered "Pentothal Sodium" intravenously to 30 patients without ill effect. Tisdall showed that the amount of sulphur ingested in twenty-four hours in bread and in eggs would be about the same. It is illogical to exclude eggs and allow bread. On the other hand, it has been shown that sulphur uncombined might be a source of danger. Niblock gave a normal invalid diet (including eggs and onions) and administered sulphates as required to 100 patients receiving treatment by one or other of the sulphanilamide group. Cyanosis was noted in three cases only. It was unaffected by the administration of methylene blue or glucose. No abnormal spectroscopic bands were observed in the examination of the blood. One of the three patients was an old man suffering from cardiac failure. No cause is given for the cyanosis in the other two. It is noteworthy that the drug used was sulphapyridine in all of these three cases. Niblock advises against the use of quinine sulphate, as in his experience it causes excessive vomiting. The frequency of cyanosis in this series is considerably lower than experience in Australia would lead one to expect. It may be that minor degrees of cyanosis were missed in Niblock's dark-skinned patients.

[1] *The Indian Medical Gazette*, August, 1941.

Abstracts from Medical Literature.

PÆDIATRICS.

Primary Infantile and Juvenile Pneumonia.

MOUNTJOY ELLIOTT (*Archives of Disease in Childhood*, June, 1941) has reviewed 895 cases of infantile and juvenile pneumonia in Dublin for the years 1937 to 1939. The age period ranged from a year and under to twelve years. All patients dying within twenty-four hours of admission to hospital or in whom signs of improvement had set in before treatment was started were excluded. The cases were clinically divided into two groups of lobar and atypical pneumonia. The former showed clinical evidence of lobar consolidation, the latter irregular patchy consolidation. The age of the patient and the pathological type of causative organism are still of great importance in determining prognosis. There appears to have been a lowering of the death rate from atypical pneumonia in the first year of life since the introduction of chemotherapy, but the results in the treatment of lobar pneumonia in infants by chemotherapy alone are disappointing. Statistical and experimental studies suggest that the administration of serum with chemotherapy in infantile pneumonia gives the best results. Too much reliance should not be placed on chemotherapy alone without an effort to better the developing immunity mechanisms by the giving of serum. The antipneumococcal immunity mechanism is relatively weak in infants. Children over two years of age with pneumonia can be treated in a similar manner to adults. All infants under two years of age with pneumonia should be treated in a properly equipped hospital. Blood transfusion, parenteral administration of fluid, oxygen, sedatives and skilled nursing are almost as valuable as chemotherapy in severe pneumonia in infants. The importance of the maintenance of weight during treatment is emphasized; efforts to continue breast feeding should be made.

The Treatment of Infantile Tetany with Dihydrotachysterol.

THERESA T. WOO, CHUAN FAN AND FU-T'ANG CHU (*The Chinese Medical Journal*, August, 1941) report their clinical experience in the oral administration of an oily 0·5% solution of dihydrotachysterol to five Chinese infants suffering from tetany. They consider that a congenital lack of vitamin *D* and calcium is an important factor in these cases. Dihydrotachysterol was highly effective in controlling the condition in four out of five cases. Fairly large doses, 1·0 to 1·5 cubic centimetres daily for one to four days, were necessary to raise the serum calcium content to a normal level. The authors state that Albright and his associates concluded that the fundamental actions of vitamin *D* and dihydrotachysterol are similar: an increased absorption of calcium from the intestine and an increased excretion of phosphorus in the urine, the ratio of the latter to the former being greater with dihydrotachysterol. The increase of calcium and decrease of inorganic phosphorus in the serum are secondary to these fundamental actions. The chief advantage of dihydrotachysterol in the treatment of infantile tetany appears to be its prompt action; the principal objections are its high cost and the danger of hypercalcæmia if it is used in excessive doses.

The Clinical Use of Vitamin K.

RAÚL ORTEGA (*Boletín-Médico-Social de la Caja de Seguro Obligatorio*, May-June, 1941), in a survey of the recent literature on hæmorrhagic disease of the newborn, arrives at the following conclusions: (i) In the newborn there is a definite hypoprothrombinæmia, which is more pronounced in premature babies; this condition reaches its maximum intensity between the second and fourth days of life—the period when hæmorrhagic disease of the newborn most often appears. The reason for this hypoprothrombinæmia is obscure. (ii) The regularity with which hypoprothrombinæmia occurs in the newborn, and the fact that this state accompanies either hæmorrhages or potential hæmorrhages indicates the usefulness of routine tests of the time of prothrombin formation; it is imperative before any surgical procedure undertaken in the first days of life. (iii) Administration of vitamin *K* raises the prothrombin content of the blood, prevents hæmorrhages or cures them if already present. (iv) Administration of vitamin *K* to the mother some hours *ante partum*, although without effect on the state of the maternal blood, raises the prothrombin content of the baby's blood to about three times its level, thus acting as a prophylactic in hæmorrhagic disease of the newborn. For this reason the routine prophylactic administration of vitamin *K* to mothers is recommended. Dramatic results are claimed in 18 cases of obstructive jaundice in which patients were treated with extract of powdered cod-fish mixed with bile; the diminution in formation time of prothrombin was greatly reduced. Rarer conditions, in which, it is claimed, vitamin *K* has been used with success, are *icterus gravis*, *anæmia neonatorum* and *hydrops fœtalis*.

Hæmorrhagic Disease of the Newborn.

LENDON SNEDEKER (*The Journal of Pediatrics*, July, 1941) presents data on 358 cases of hæmorrhagic disease. The data presented substantiate previous observations on hæmorrhagic disease, the term now generally reserved for cases in which hæmorrhage is spontaneous and idiopathic. The author feels justified in discarding certain ætiological theories. It is rare to find in the affected organ a definite bleeding point, ulceration or other focal lesion accounting for the hæmorrhage. Intracranial hæmorrhage does not consistently accompany hæmorrhage from the intestinal tract. The low incidence of prematurity and the normal development shown by these children afford indirect evidence against the theory that their blood vessels are immature and therefore more subject to the effects of trauma and asphyxia. It still remains to be demonstrated that there is some specific structural defect in the blood vessels. The increased incidence of asphyxia may be of ætiological importance. While asphyxia is not a consistent factor, one suspects that it frequently initiates hæmorrhage or in other cases brings out a latent hæmorrhagic tendency. It is, however, only one of the possible factors responsible for the hæmorrhagic syndrome. Transfusion appears to be of definite value in treatment. A fatal outcome is more likely in infants having asphyxia or intracranial or "internal" hæmorrhage, and the results reported by any observer will depend on the frequency with which he observed these more serious forms of hæmorrhagic disease. In the author's series the increased mortality encountered in untreated cases was apparently not due to such factors. There are a number of reports in the literature commenting on the remarkable rapidity with which hæmorrhage has been seen to stop following transfusion. It may be that the child receives benefit merely by the replacement of blood, but the amount given by vein is frequently quite small in proportion to the quantity of blood lost, and the results are dramatic nevertheless. Neither does it seem likely that the effects of asphyxia, trauma or infection could be so quickly offset. There are no data in any of the author's cases on the quantitative level of prothrombin. Present knowledge does not permit one to say categorically that these children regularly suffer from such a defect in the blood-clotting mechanism. Evidence accumulating in the literature substantiates the hypothesis that there is very probably in some cases a prothrombin deficiency which can be relieved by the administration of vitamin *K*, and that this drop in the prothrombin level occurs at least to a slight degree in most newborn infants. Normal prothrombin levels will bear further study. The evidence does not at the present time justify the conclusion that this prothrombin deficiency is a constant finding or that patients with hæmorrhagic disease can be treated satisfactorily by the administration of vitamin *K*. Until such proofs are available, transfusion should continue to be the treatment of choice in hæmorrhagic disease. Unless the infant shows evidence of considerable blood loss, 50 cubic centimetres are usually an adequate amount. In a small percentage of cases more than one transfusion will be required. In regard to a choice of donor, it is apparently immaterial whether the mother, father or some other person is used. It is wise during the first week of life to refrain from any manipulation or operation likely to cause bleeding, as fatal bleeding has been known to follow such procedures as circumcision or cutting the frenum for tongue-tie.

Favism in Children.

P. ROBINSON (*American Journal of Diseases of Children*, October, 1941) reports from Tel Aviv observations on a disease which seems to have been hitherto unknown in Palestine. The disorder is favism, an acute hæmolytic anæmia due to the ingestion of broad beans (*Vicia fava*). Favism is not uncommon in Sicily and Sardinia; only sporadic cases have occurred in other countries, for example among Italian immigrants in the United States. The onset of the illness is sudden. The first symptoms are vomiting and diarrhœa. The urine is reddish-brown; the skin turns pale and grey, and the eyes become sunken and consciousness clouded. The number of erythrocytes may fall to 1,000,000 or less per cubic millimetre, and the hæmoglobin content to 10%. Recovery, which begins spontaneously, in general is assisted by blood transfusions and takes one to two weeks. Fatalities are rare. The

pathogenesis of the disease is obscure. It occurs not only after the ingestion of uncooked or cooked beans, but as a result of inhalation of the blossom dust. This and the marked eosinophilia in both blood and bone marrow support the allergic theory. Examination of the bone marrow suggests that there is a stoppage of the production of red blood cells during the hæmolytic period and excessive production during the first days of recovery. The course of the illness resembles that of Lederer's anæmia, but in favism relapses are common. The authors observed six patients with the disease, and three of them had two attacks. They consider that the disease is more common than is supposed, and suggest that some of the patients with so-called black-water fever may in reality suffer from favism.

ORTHOPÆDIC SURGERY.

Severe Supracondylar Fractures of the Elbow.

EDWIN M. MILLER *et alii* (*Annals of Surgery*, June, 1941) describe a form of treatment carried out on 82 children suffering from supracondylar fractures of the elbow. In the 74 cases observed long enough to establish accurate end results, the outcome was good except in two instances. The deformity, in most cases, is a displacement of the short lower fragment backwards with inward rotation, together with lateral or medial displacement. The long upper fragment is anterior and its sharp edge may injure the median nerve, or may even pierce the skin. The authors' method is to apply traction in four directions to reduce the deformity, and to maintain traction for three weeks with the patient in a recumbent position, until union is firm. A hole is first drilled in the ulna, one inch distal to the olecranon. An eyelet screw is turned securely into the bone. The child is placed in a crib and the arm, flexed to a right angle, is passed between the padded bars of the crib. A widely spread Thomas splint is attached to the crib, and over its cross bar traction is applied by means of a cord and a weight of about five pounds. Upward pull on the lower fragment is procured by the application of adhesive strapping to the forearm, and the attachment of a three-pound weight to a traction cord. Downward pull on the upper fragment is obtained by a weight of three pounds, suspended from a well-padded sling. Skiagrams are taken, and minor adjustments of the lines of traction or variation of weights are sufficient to obtain good alignment.

Osteitis of the Metatarsal Sesamoid.

RODNEY SMITH (*The British Journal of Surgery*, July, 1941) discusses the incidence of metatarsal sesamoid pain and includes a report of a case of acute pyogenic osteomyelitis of one of these bones. The metatarsal sesamoid rarely exhibits the pathological lesions which are common in other bones. Pain in the sesamoid is not uncommon, although the pathology of the condition is obscure. Osteochondritis, sesamoiditis and Köhler's or Schlatter's disease of the sesamoid are among the names applied. The radiological appearance is one of stippling attributed to areas of necrosis in the marrow and trabeculæ. It is pointed out that the metatarsal is often normally divided into two or more ossicles. Schutz gave it as his opinion that only in the presence of this division does the pathological condition arise. Inge and Ferguson disagree with Schutz, and after a study of a series of cases of sesamoid pain come to the following conclusions: (i) fracture of the sesamoid is very rare; (ii) the actual bony changes of Köhler's or Schlatter's disease are also rare. In most cases the pain is due to bursitis, luxation or arthritis. Acute pyogenic osteomyelitis of the sesamoid is extremely uncommon, and only two authenticated cases and two doubtful cases have been recorded. The author's case occurred in a girl of thirteen with a history of trauma to the ball of the foot five months before. X-ray examination revealed a tripartite sesamoid, but no radiological evidence of osteomyelitis became evident until a sinus, discharging pus, appeared on the dorsum of the foot. Subsequent X-ray examination revealed complete detachment of the lateral sesamoid which had become displaced proximally. The sinus was laid open, the necrosed sesamoid removed and the track lightly packed with ribbon gauze. The patient rapidly recovered.

Coxa Plana.

H. JACKSON BURROWS (*The British Journal of Surgery*, July, 1941) discusses the pathology and nature of *osteochondritis juvenilis*. He considers the condition from clinical and radiological examinations of osteochondritis of the femoral head and by direct examination and microscopic study of similar disease in other bones. *Coxa plana* occurs between the ages of two and eighteen years, and three-quarters of the patients are boys. In about 10% both hips suffer, though almost never at the same time. A history of trauma varies in different statistics from 14% to over 50%. Examination of the second or third metatarsal head in Freiberg's (or Köhler's second disease) and semilunar bone affected in Kienboch's disease, which are described as similar pathological conditions to *coxa plana*, show that the outstanding features are: (i) necrosis of the bone with lysis of the trabeculæ; (ii) invasion by young connecting tissue resulting in (*a*) phagocytosis, (*b*) regeneration. The chief destruction is situated subchondrally and the term "subchondral lysis" is suggested as better than "subchondral necrosis", the term usually applied. The writer advances an explanation of two processes responsible for the radiological changes seen in these cases: (1) The increase in the joint interval above the epiphyseal nucleus is due to increased thickening of the femoral articular cartilage. The usual explanation is that the appearance is due to failure of bony invasion of the cartilage by the epiphysis. (2) Flattening of the epiphyseal nucleus probably depends upon destruction within the nucleus, since the author has found in early cases that there is no increase in width of the nucleus. (3) Rarefaction is the result of destruction of trabeculæ and their replacement by transradiant necrotic débris. Later, the ingrowth of young phagocytic and reparative tissue may be a factor. (4) Increased density in the epiphyseal nucleus is probably due to several factors, among them being calcification of débris. The radiological and histological appearances in osteochondritis are contrasted with those found in aseptic necrosis. Example of the latter are found in fractures of the carpal navicular and femoral neck. In aseptic necrosis the bone trabeculæ show increased density and the texture is perfectly preserved except when secondary collapse supervenes. The most striking histological change is death of the marrow. In osteochondritis bony texture is blotted out at an early stage, and this destruction precedes the invasion of young phagocytic and reparative tissue. The cause of the difference between the two processes may well depend upon the special circumstances common to such situations as the epiphysis of a child and the bony centre of a fully developed bone in an adult. Both conditions may possibly result from arterial anæmia, but in the difference of anatomical structures referred to the effects of this are atypical. Numerous attempts at experimental production of *coxa plana* in animals have failed to reproduce the radiological and histological characteristics of this condition. Treatment aims at the maximum preservation of the femoral head, and functional prognosis is best in the young. The logical ideal in eliminating body weight and muscle pull is continued traction until healing has occurred, a matter often of years. The most generally suitable treatment is still the use of the weight-relieving caliper. Whitman's operation is recommended only when the condition starts late, at about the sixteenth year. In this uncommon event the response to conservative treatment is often bad.

Synovectomy of the Knee Joint.

THE end results of synovectomy of the knee joint are analysed by Ralph K. Ghormley and David M. Cameron (*The American Journal of Surgery*, September, 1941). The procedure was first described in 1922 by Swett. Since then the operation has become accepted as of value in certain types of joint conditions. Key's experimental work on rabbits' knee joints, in which the joints were examined microscopically at intervals of from two to one hundred and four days after operation, shows that in these animals, the synovial membrane was normally regenerated within sixty days after synovectomy. The new synovial membrane is formed by metaplasia of underlying connective tissue. Swett recommended that the operation be performed only in cases of chronic infective arthritis. A study of results obtained from Steindler's clinic indicate that in cases of chronic polyarticular arthritis 60% of patients were improved by synovectomy, whereas in the monarticular types 75% were improved. In traumatic arthritis 95% were improved. Synovectomy is obviously best suited to the knee joint. The surgical approach to this joint is excellently adapted for extensive removal of the synovial membrane. The operation may be carried out on the elbow or wrist, but complete synovectomy is unlikely to be obtained. The hip and the shoulder are almost unapproachable for such a procedure. The authors present in table form a series of 88 patients on whom synovectomy was performed at the Mayo Clinic. Their conclusions are that the procedure is most useful in traumatic arthritis, synovial osteochondromatosis and xanteroma or benign tumours. They consider that the operation is indicated, though of less value, in chronic infective arthritis. It is of value in chronic synovitis, but the prognosis in these cases must be guarded; this is also true of intermittent hydrarthrosis.

British Medical Association News.

SCIENTIFIC.

THE third annual general and scientific meeting of the Ophthalmological Society of Australia (British Medical Association) was held in the Lecture Hall of the Royal Australasian College of Surgeons, Melbourne, on October 15, 16, 17 and 18, 1941, DR. J. C. MITCHELL, the President, in the chair. Those present included two members from Queensland, two from Tasmania, one from Western Australia, three from South Australia, seven from New South Wales, and twenty-five from Victoria.

President's Address.

The President extended the welcome of the Society to such guests as had been able to attend, and noted with gratification the presence of the Society's first President, Sir James Barrett, and its second President, Dr. James Flynn, whose year of office was spent abroad with the Royal Australian Navy.

He then welcomed members of the council of the Society and all the visiting delegates, stressing the importance, especially in time of war, of such contributions to urgent problems as might be put forward at a truly representative gathering of Australian ophthalmologists. Referring to the wide distribution of the last *Transactions* of the Society, the President thanked those responsible for the compilation of the volume.

The President then offered the sincere sympathy of the Society to the wife and family of Dr. Zelman Schwartz, who was killed in action at Tobruk. The meeting rose and stood in silence in honour of their gallant colleague.

After thanking the council and members of the Society for allowing him to be their President and for loyally supporting him in that office, the President then traced the growth of several ophthalmological societies and the formation of the present Federal body. Exhorting members to carry out to the full their responsibilities as medical practitioners and emphasizing the need for meticulously careful and unhurried work, he then briefly reviewed some of the more modern trends in ophthalmic medicine and surgery, including the following subjects: refraction, mydriasis, antisepsis, anæsthesia, cataract operations, instruments, detachment of the retina, visual fields, sulphonamides and glaucoma operations. Also he referred to the important question of visual standards, which, he said, should be treated from an Australian rather than from a State or individual viewpoint.

Concluding his address, the President pointed out that all lived in changing times and that it behoved the medical profession to fit in with such readjustments as were necessary for the common weal.

Finally he reminded members of their great obligations and responsibilities, and of the spirit of the oath of the father of medicine, the great Hippocrates.

The Cause of the Decline of Trachoma in the Southern Portion of Australia.

SIR JAMES BARRETT (Melbourne) read a paper tracing the remarkable decline in the incidence of trachoma, from the time he began practice in 1888 to the present time. At one time the commonest and most harassing of diseases, trachoma had gradually and progressively lessened in severity and incidence. As all the new cases seemed to be coming from the country, in 1909 Sir James Barrett, Dr. Orr and Dr. Lynch set out to make a survey of all trachoma patients seen, with the object of trying to find the place of origin of the disease. Victoria was divided into districts by the investigators, and the average temperature and humidity in these districts were compared with the infection density in such areas. Broadly speaking, infection was most prevalent in conditions of heat, dryness, dust and low humidity.

Sir James Barrett referred to the evidence of Lieutenant-Colonel Wright, of Madras, whose experiments in implanting trachomatous material seemed to show that the disease was not ordinarily very infectious. Evidence was brought forward to show that many other factors (including food, santitation, general hygiene and nutrition) could not be regarded as causative of themselves alone, though they all came into the picture. The disease was still met with west of the wheat belt in New South Wales and Queensland, but in southern Australia it was a disappearing disease. Despite the prominence of the climatic factor, Sir James Barrett said that his own investigations, at any rate, pointed to the existence of some factor or factors yet to be determined.

The Refraction of an Urban Group in Australia, with Comments on Myopia.

DR. E. L. GAULT (Melbourne) read a paper analysing the refraction of the eyes of all patients examined by him in private practice during the period 1905 to 1934 inclusive. The visual acuity was recorded while the eye was under the influence of homatropine and cocaine. The number of eyes examined was 31,809. Of these, 39·7% were emmetropic, 44·1% hypermetropic and 16·2% were myopic. Dr. Gault agreed with the view that the different grades of refraction represented variations tending to an optimum at the near emmetropic level, and having upper and lower limits which might be considered to be within physiological bounds. A relatively small group, including myopes of over 10 dioptres, were outside these bounds. The causes operating to produce the varying refractive conditions were discussed. It was suggested that myopia over 10 dioptres had a congenital origin. This analysis provided the following evidence in support of this theory: (i) the high proportion of unilateral high myopia or anisometropia of high degree, (ii) the presence of high myopia in very early life, (iii) the association with gross visual defects, and (iv) the presence of a posterior staphyloma in a large majority of these highly myopic eyes. The treatment of young myopes was discussed. Dr. Gault had found it sufficient to forbid, at most, home reading and to encourage outdoor occupations and sport. If the myopia continued to increase at the rate of one dioptre a year Dr. Gault had usually sent the child to a farm or station for a year, forbidding all near work. He had found that medium and even high myopia was not incompatible with the successful prosecution of a clerical calling. This subject was one of five included in a *questionnaire* that had been sent to oculists prior to this meeting. The replies received were summarized. Extensive tables and a graph were exhibited.

Modern Trends in Illumination.

MR. L. D. WRIGHT, A.M.I.E. (Aust.), read a paper and exhibited slides dealing with modern practice in illumination. He stated that war-time industrial conditions had brought a realization of the appalling inefficiency of most industrial lighting. In other countries the medical profession had given a lead in regard to lighting standards. Mr. Wright asked Australian ophthalmologists to help illuminating engineers in their problems. He then discussed the amount of light, the quality of light, the relation between illumination and visual acuity and the problem of "glare", giving figures as to the number of foot-candles necessary for various types of work. In summing up from all his data, Mr. Wright brought forward the principle of a large quantity of evenly distributed light as the ideal, in contradistinction to intensely bright individual sources of light, which caused glare, shadows and excessive contrasts. The best means of obtaining this objective would seem to be fluorescent lighting (an electric discharge through a gas) as it had a high efficiency, low radiant heat and was admirably suited to the method of long tubular light sources.

Discussion on Orthoptics and Aviation.

DR. A. K. MCINTYRE and DR. S. MORSON reported on their investigations into the problem of whether the extrinsic ocular muscles possessed proprioceptive sensibility. Their evidence included previous work on the subject, anatomical and histological descriptions, physiological considerations, experimental results and observation of various ocular phenomena. Their final conclusion was that the ocular musculature did not possess proprioceptive sensibility. Illustrations and gramophone records were used to demonstrate their work and results.

SQUADRON LEADER A. L. TOSTEVIN (Adelaide) read a paper commenting on statistical information obtained by Miss Willoughby and Flight-Lieutenant Pridmore, relative to the effect of orthoptic defects on pilot performance. He stated that the number of cases in which ocular defects were the primary cause of the failure of a pupil trainee was negligible. Convergence deficiency did appear to be a handicap, but pupils satisfactory in other ways rapidly learned to overcome their difficulties from this source. However, the work done had shown the necessity for further research, done in such a way that figures could be completed and results shown. Accordingly a scheme had now been evolved for Parafield, whereby half of all pupils with ocular defects would go through without any orthoptic training, the other half being treated for their defects. Some standard had to be set up and it would appear that two factors relative to the phorias needed estimating: (i) the degree of energy demanded of the subconscious mind to maintain binocular function under certain conditions; (ii) the state of mental, physical and ocular muscle fatigue that was induced by this persistent effort.

MISS L. WILLOUGHBY read a paper giving details of research into the effects of ocular conditions on pilot training. Miss Willoughby began by pointing out that orthoptic training had been very successful in bringing many men with ocular defects up to the standards for Royal Australian Air Force acceptance.

With these results in mind, a survey of the detailed state of the binocular vision of pilots in training at Number 4 Elementary Flying Training School was made. The visual classifications obtained were compared with the performance of the pilots. Contrary to expectations, the statistical evidence obtained did not show that men with the best orthoptic standards were necessarily, or even usually, better pilots than those with poorer orthoptic results. Miss Willoughby gave tabulated figures illustrative of these conclusions, but said that despite all this, one felt that eyes in a perfect state were more likely to stand up to conditions of continuous strain. Her statistics dealt mainly with range of convergence, voluntary convergence and depth perception (stereopsis).

WING COMMANDER W. D. COUNSELL (Hobart) pointed out that recent developments in aircraft design had not lessened the need for accurate visual judgement in flying. If orthoptics could help this judgement, it was a particularly important subject at present, when man power was limited and time was a factor. The visual tasks set by flying were now better appreciated, but the manner of their fulfilment was still insufficiently understood. To assess the value of orthoptics both theory and practice must be considered. Wing Commander Counsell outlined theoretical aspects of visual judgement and discussed the elements of depth perception. He said that the binocular elements were most likely to be helped by orthoptics. He considered the difficulty of assessing the relative value of the isolated factors and the importance of completeness and meaning in the visual presentations.

An apparent lack of precision in the fixational movements of the eyes was found despite higher accuracy in the final percept. Care, therefore, should be exercised in attaching significance to small deviations in apparently normal men.

Wing Commander Counsell went on to discuss the relationship of inaccuracies of judgement to manifest faults, and made particular reference to the problem of the variable effect of heterophoria and to possible fallacies in assessing by stereometric tests a pilot's ability to estimate height. He stressed the importance of attention in visual perception. He said that many other requirements, which later became automatic, divided the attention of the pupil. Orthoptics, by facilitating visual perception, might help the pupil over his most difficult period. At present ophthalmologists did not properly know what they were doing in orthoptics, and further research on the visual mechanisms was necessary. The application of orthoptics to aviation differed from its clinical use. Its value in the former might be assessed by individual improvement in flying performance and by mass statistics. The value of orthoptics in squint was still debated, but the published opinions of authorities on aviation medicine were quoted as being generally favourable. The results of the present work in the latter field were, therefore, of great interest.

DR. T. A'B. TRAVERS (Melbourne) reported having undertaken extensive investigations of the fusion amplitude of trainee pilots at an initial training school. The results obtained bore little or no relationship to the flying ability of the pilots tested.

DR. RINGLAND ANDERSON (Melbourne) summarized the results that a team of workers had obtained during the last year at an elementary and a service flying training school. They found that stereoscopic vision and the cortical control of convergence were defective in many trainees on arrival, but that brief training was adequate to develop full powers in the great majority. There appeared to be a definite relationship between such visual and ocular defects and flying ability. Two criteria were adopted: (*a*) the time taken by a man to fly solo in comparison with the average time for his group of trainees who would be learning under almost similar conditions; (*b*) the report on his early and later flying as given by his flying instructor. It was found that men with weak stereopsis tended to take longer than average to fly solo and to have bad initial reports. The importance of controlled convergence was shown in several tables. Most trainees without this power had slower solo times, more failures and worse early reports than those with it. Its value appeared to be greatest in those with esophoria. The influence of fatigue as a cause of excessive convergence was emphasized. Its treatment by relaxation was described. The orthoptic methods known in civilian practice had been modified, and with experience it was possible to obtain results more quickly than a year ago. This meant that the benefits from these results were available at an earlier stage in a pilot's training than formerly. The influence of this was shown by a comparison of a recently trained group of 150 trainees with the first trained group of similar size. Dr. Anderson considered that orthoptics was as necessary for modern pilots as physical training was for the infantry.

Orthoptics in General Examination of the Eye.

In the absence of DR. JOHN MAUDE (Sydney) his paper was read by DR. ARTHUR D'OMBRAIN (Newcastle). The paper began with a reference to the increasingly wider application of orthoptic training. Then the subject of muscle efficiency was emphasized; some patients with great exophoria had no symptoms, whereas others with small phorias had pronounced symptoms. Dr. Maude stated that this apparent anomaly was explicable in terms of convergence fatigue. Dealing with suppression, he pointed out that if stereoscopic vision failed, suppression resulted; if convergence failed, again suppression was the result. But even with perfect stereoscopic binocular vision there existed a physiological suppression, to enable the subject to avoid the double vision of objects beyond the point of gaze; it was this physiological suppression that made it easy for pathological suppression to follow readily on faulty balance of the horizontal muscles. The constant association of exophoria with hyperphoria was explained by the statement that the retreat from discomfort by way of suppression in the vertical axis was closed and must be sought for in the horizontal axis; the first step towards suppression in this axis was found in exophoria. Convergence disabilities should be treated by an orthoptist or, failing this, by the home use of the amblyoscope.

Some Observations on Intraocular Pressure.

DR. T. L. MCLARTY (Adelaide) reviewed some theories of the formation of the intraocular fluid (aqueous). The earlier observers favoured a theory of secretion, though some considered a process of simple transudation sufficient to explain phenomena. Duke-Elder had developed work done by Magitot, Baurmann and Messman, and had elaborated a theory of dialysation. Dr. McLarty discussed evidence for and against the dialysis theory and went on to describe experiments carried out in Adelaide by himself and co-workers, with what they believed to be a superior technique and apparatus. He then described in detail the apparatus and the technique used in experiments both upon the intact animal and the perfused head, and exhibited results showing the variation in arterial, venous and intraocular pressures following hæmorrhage, and the variations in intraocular pressure when the osmotic pressure of the blood was altered by transfusion of isotonic and anisotonic saline solution. From his results, based upon observations of experiments on hæmorrhage followed by transfusion and on simple transfusion combined with the effects observed in the perfused head, Dr. McLarty said that it would seem evident that the aqueous was not formed by simple dialysis.

Physical Therapy in Eye Diseases.

DR. FRANK MAY (Melbourne) read a paper in which he discussed the use of physical therapy in eye diseases; he made particular reference to a series of fifty cases in which short-wave therapy had been used. He reviewed heat therapy in its various forms. Dr. May distinguished far infra-red from near infra-red therapy; he stated that there were some dangers in the use of near infra-red radiations and no particular advantages. Ultra-violet light could also be divided into near and far groups, the far rays, from 2,900 to 2,000 Ångström units, being the more active. Ultra-violet rays were used in two ways: as a general tonic stimulant and locally for ulcers of the lids and cornea. Dr. May stated that the best method of heat application to ocular structures was the use of short-wave therapy; he gave details of technique and the indications and contraindications for short-wave treatment. Finally he reviewed the results obtained during the last year with this treatment in some fifty cases. Results were very encouraging.

Congenital Cataract associated with Maternal German Measles.

DR. NORMAN MCALISTER GREGG (Sydney) read a paper dealing with the occurrence, during the first half of 1941, of an unusual number of cases of congenital cataract. The total number of cases included in the author's review was 78. In his detailed consideration of these cases Dr. Gregg dealt with the peculiar characteristics of the cataracts themselves, with associated conditions in other organs, with ætiology, distribution, treatment and prognosis. Special points about the cataract itself were: the marked density and large size of the opacity, the difficulty in obtaining

mydriasis and the high frequency of intolerance to atropine. The most striking associated condition was the presence, in 46 cases, of a congenital defect of the heart. When cases continued to appear, both in his own patients and in those of many colleagues, Dr. Gregg had turned his attention from the clinical condition itself to the question of ætiology, as he felt that there might be some common causal factor. As a result he had discovered an almost constant association of maternal German measles in early pregnancy, the percentage proportion being over 90.

The embryology of the lens was considered and the theory put forward of a possible infective or toxic process, resulting in a partial arrest of development. Special difficulties in management and operation were discussed. Finally Dr. Gregg asked that in all prenatal clinics and maternity hospitals careful investigations should be made (and recorded) of the exposure of the mother to infection of any kind during the entire period of pregnancy; prophylaxis was the only sure treatment. This contribution was discussed in this journal in the issue of December 6, 1941.

An Analysis of 760 Cases of Cataract Extraction.

DR. ADELAIDE GAULT (Melbourne) read a paper giving a report of 376 intracapsular extractions by Smith's method from Sir Henry Holland's Skirarpur Clinic in 1941. The pre-operative and post-operative treatment and the technique of the operation were described. The complications and the contraindications were summarized. The results obtained were compared with the remainder of the cases in which extraction with a capsulotomy was performed. The vitreous loss, chorioidal hæmorrhage and iris incarceration were less frequent after the latter operation. The immediate visual result was much better, however, after the intracapsular method. Dr. Gault was very impressed with the results obtained by this method in the skilled hands of Sir Henry Holland.

Discussion on Night Vision.

SURGEON LIEUTENANT-COMMANDER A. JAMES FLYNN (Sydney) read a paper in which he stressed the importance of night vision in the Navy in war-time. He described the construction and use of an adaptometer, a device testing the extent of dark adaptation. The adaptometer described gave only comparative findings, but these were of great value in classifying the night vision of personnel. The author had tested 332 of the company of an Australian cruiser with the instrument. A short summary of his figures showed 126 above average, 116 average and 90 below average. It would be of value to test the same group for rate of dark adaptation after exposure to bright light. The use of a silhouette in the test was of practical value.

DR. H. GAULT (Melbourne) reported results after measuring the night vision of 850 men at a naval training depot. He emphasized the value of controlling the illumination with a voltmeter and photometer. Some bad results were attributable to stupidity. Men with subnormal night vision would be unsuitable for night lookout duties.

PROFESSOR R. DOUGLAS WRIGHT (Melbourne) pointed out that absence of vitamin *A* was one cause of night blindness. The deficiency could arise from defective supply or choice of food or from defective alimentary absorption. Proof of vitamin *A* deficiency consisted in the manifestation of a marked improvement in dark adaptation on altering one condition only, namely, the supply of the vitamin. There was only scanty evidence as to whether high feeding with vitamin *A* could improve night vision, but one observer had stated that the light threshold could be reduced to one-half by overfeeding with the vitamin. Finally, Professor Wright pointed out that vitamin *A* deficiency could result in an irreversible degeneration of the cone mechanism of the retina. The irreversibility of this cone change made imperative the prevention of vitamin *A* deficiency.

WING COMMANDER W. D. COUNSELL (Hobart) said that night fighting had increased the importance of good night vision in aviation. He quoted night values involved and compared them with the figures for familiar illumination; a clear moonless night provided an illumination of about 0·0002 foot-candle. The visual elements employed required separate measurement and were of varying importance for differing purposes. Certain variables in test and subject had to be considered in the making of an accurate test. Some speed in adaptation was desirable for night flying when looking from instruments to sky. The recognition of other aeroplanes required good form sense. The psychological factors associated with this made an accurate scientific measurement of light minimum or revolving power, inadequate for the purpose of selecting air crews for night work. Thus the problem of designing a suitable testing instrument presented special difficulties. One such instrument would be demonstrated by the next speaker.

SQUADRON LEADER J. L. R. CARTER (Launceston) said that the apparatus which he showed was originally introduced and designed by Group Captain Livingstone, consultant in ophthalmology, Royal Air Force. It was an attempt to obtain from one examination an indication as to a candidate's ability to see in the dark, his psychological make-up and his potential suitability for night flying. Prior to the test each candidate wore a pair of dark goggles for thirty minutes. He was then taken into an optically dark room and seated three feet from the panels of the hexagon; the goggles were removed and he remained in complete darkness for a further fifteen minutes. The panels, which contained four rows of letters with three letters in each row, were next uncovered. They were then dimly illuminated by 40% of the transmitted light and the applicant then wrote down what he saw on a block of paper. One minute was allowed. The hexagon was rotated, illumination was increased 10% and another minute was allowed to examine the new panel and record the letters. It was once more rotated with a further increase in illumination by 10%. The applicant was next light-adapted for one minute; the room was again blacked out and a new panel was shown. A system of marking recorded his results on a percentage basis. An average figure was arrived at after a period of experimentation, and candidates were graded as excellent, above average, average, below average and poor. Six candidates could be tested at each session, which was of one hour's duration, so that forty-eight could be put through in an ordinary eight-hour working day.

Discussion on Colour Vision and the Services.

DR. W. CHRISTIE (Adelaide) read a paper giving a summary of prolonged and varied observations, made with various tests, into colour vision and its defects. By colour blindness Dr. Christie meant a defective discrimination of colours, not necessarily the invisibility of such colours. The principle of discrimination was the basis of the pseudo-isochromatic tests of Stilling, Ishihara and Edridge-Green. Dr. Christie gave details of his classification of the colour blind, ranging from a "Nought Group" to the "Seven Unit" class.

Colour blindness might differ in the two eyes and monocular colour vision existed. Reexaminations often showed variations in spectral readings, but when Ishihara's test was used the variations were very slight. Fatigue appeared to be a factor in causing variation.

Dr. Christie suggested a period of provisional enlistment for recruits whose colour vision was doubtful; he considered the present signal colours the worst that could be used, blue and yellow being preferable to green and red; also he recommended the experimental use of vitamins *A* and B_1 in cases of subnormal colour vision.

DR. DONALD BUCKLE (Newcastle) read a paper dealing with the outlook of the medical psychologist towards colour vision. Dr. Buckle's paper consisted, first, of a short review of some of the work done by Gestalt psychologists concerning colour vision, including colour constancy experiments, the distinction between surface and film colours, effect of colour not in the field of visual awareness; and, secondly, of findings in relation to the emotional effects of colour as demonstrated in the Rorschach ink blot method of personality examination, both in normals and in the colour blind. The induction of colour blindness by hypnosis was also reviewed. While touching on the theoretical implications of the phenomena described by him, Dr. Buckle was most insistent on the profoundly practical importance of the emotional effects and disturbance of performance produced by the perception of colour in ordinary conditions. He considered that the ophthalmologist, being the acknowledged authority on the physical aspect of colour vision, should be just as much interested as the medical psychologist in the phenomena discussed.

Irido-Ciliary Melanoma of an Only Eye Treated by Radon.

DR. LEONARD MITCHELL (Melbourne) gave a report on a patient, aged sixty-one years, whom he and Dr. Kaye Scott had treated with radon. A large black mass was seen invading the root of the iris. The vision was 6/36 and the tension was normal. Five radon seeds were fixed to the sclera by silk sutures and left *in situ*. A larger seed shaped to the curve of the limbus was left in position for five days and then withdrawn. A cataract developed, which would be extracted later. The visible portion of the tumour had almost disappeared.

Tasmanian Blindness: A Final Survey.

In the absence on active service of DR. J. BRUCE HAMILTON (Hobart) his paper was read by DR. J. RINGLAND ANDERSON (Melbourne). As Vice-President of the Society, Dr. Hamilton deeply regretted his inability to attend the meeting; he wished the meeting every success under the presidency of Dr. Leonard Mitchell. His paper revealed the incidence of binocular and monocular blindness in 6,770 cases examined in private practice in Tasmania. A comparison of these figures with those reported by J. Ringland Anderson (1939) in Victoria was given. The paper also again stressed Dr. Hamilton's opinion that all men enlisted with vision of less than 6/9 should have a complete ophthalmological examination, and that all with defective vision should be provided with spectacles in duplicate, one pair in gas-mask frames. Twenty-six analytical and comparative tables, dealing with the main subject of the paper, were presented and some final conclusions were drawn.

SQUADRON LEADER J. L. R. CARTER demonstrated the Giles Archer lantern. This small unit, designed by G. H. Giles and modified for Air Force purposes by Group Captain Livingstone, Royal Australian Air Force, consisted of a lamp house, a slide with three apertures and a set of filters enclosed in a disk. The filters were composed of gelatine enclosed in plane glass, and placed far enough away from the lamp to be unaffected for general use by any radiated heat. The filters were, in order, dark red, yellow, standard yellow, standard green, blue-green and light green; standard red, standard yellow and standard green approximated very closely to the specifications laid down for civil aviation by the International Congress for Aerial Navigation. The test was carried out in darkness, and the candidate was shown the complete sequence of colours through the large aperture. If mistakes were made with red and green when this aperture was used, there was no need to give further time to the test, and the candidate was colour-vision defective, unsafe. If the candidate passed with the large aperture, the second aperture was brought into view and the filters were put up out of sequence. If the candidate passed with this aperture, aperture 3 was put up and the filters were presented again.

Squadron Leader Carter described the following results.

Result A: Colour vision was normal; no mistakes whatever occurred in any filters with apertures 1 and 2. In proper conditions of darkness there should be no difficulty with any of the colours using aperture 3.

Result B: Colour vision was defective, safe; no mistakes in red and green. Standard yellow could be misnamed, and palest green misnamed white. With the small aperture all greens and reds must be correctly named; dark red could be misnamed.

Result C: Colour vision was defective, unsafe; if the candidate described red as green or *vice versa* in any sequence, he should be rejected. In the dark with aperture 3 the same mistakes would exclude him. In nearly all cases this unit confirmed the Ishihara findings. It would detect border-line candidates who passed as safe, or, in some cases, normal, with the technique employed by the Royal Australian Air Force on the Edridge green lantern.

WING COMMANDER W. D. COUNSELL (Hobart) stated that the prescribed standards for flying were to be found in the official manual (AP.130). Flying personnel must be either colour normal or possess only a slight degree of anomalous trichromatism. The tests employed were the Ishihara's plates and the Edridge-Green lantern or the Giles-Archer colour perception lantern. The speaker demonstrated a modern aviation map. Its colouring would confuse a colour defective. Air navigation and landing lights were demonstrated and the principles upon which their precise colour was defined by British standard specification were outlined.

General Discussion: Visual Standards for the Services.

LIEUTENANT-COLONEL G. H. B. BLACK (Adelaide) made a survey of Army standards of vision. Present and past standards were discussed and the ideas of members on the subject were asked for by the speaker and by the President. Other speakers then joined in the discussion both for the Army and for other services. Publication of details of this discussion is not desirable at the present time, the subject being one for the services concerned.

Contact Lenses.

In the absence of DR. JOHN MAUDE (Sydney) his paper was read by DR. ARTHUR D'OMBRAIN (Newcastle). The main part of the paper was devoted to the fitting of contact lenses. Four cardinal points were to be observed, namely: centring the pupil; perfection of scleral fit; limbal clearance and corneal contact. These four desiderata were dealt with in detail. It was pointed out that the success of contact glasses in conical cornea could be explained by the action of the cone springing the contact lens forward; the most painstaking care was necessary to ensure perfect corneal contact, either too little or too much causing intolerance. A complete account of the procedure of fitting lenses was given and difficulties were mentioned, including Sattler's veil and light intolerance. The paper stressed the need for the closest cooperation between the ophthalmic surgeon and the mechanic, responsibility having to be shouldered by the surgeon.

The Distinction between Amblyopia and Organic Defects of Vision.

DR. T. A'B. TRAVERS (Melbourne) read a paper emphasizing the importance of distinguishing *amblyopia ex anopsia* from defective vision due to organic lesions. This was of especial importance when considering the enlistment of men for air crew, as in the author's opinion minor degrees of amblyopia should not disqualify candidates. A mirror-screen test was described; it depended for its results upon the utilization of the phenomenon of suppression in amblyopic eyes. The author emphasized the fact that suppression occurred even in normal vision, one eye being in varying degree a "master eye". In amblyopia the condition of master eye was merely exaggerated.

Arcuate Scotomata in the Visual Fields associated with Injury to the Optic Nerve in the Optic Canal.

DR. KEVIN O'DAY (Melbourne) and MR. FRANK MORGAN discussed the implications of two cases showing arcuate scotomata in the visual fields. In one case X-ray examination revealed a fracture of the wall of the left frontal sinus; the other case was one of cerebral tumour with evidence of pressure on the roofs of the optic canals. In the latter case careful histological investigation by Dr. O'Day revealed that the atrophic fibres of the papillo-macular bundle rose, in the posterior portion of the optic nerve, to the dorsal surface of the nerve and maintained this position in the chiasma and that part of the optic tract examined. These findings differed from the long-accepted view that these fibres maintained a central position posteriorly. In conclusion, Dr. O'Day discussed bilateral central scotomata and stated that every effort should be made to exclude the possibility of cerebral tumour in these cases, especially when the scotomata were of arcuate form.

THE MEDICAL WAR RELIEF FUND.

THE following letter, addressed to the General Secretary of the Federal Council of the British Medical Association in Australia, from the Chairman of the Medical War Relief Fund Committee, is published at the request of Sir Henry Newland, President of the Federal Council.

British Medical Association House,
Tavistock Square,
London, W.C.1,
7th September, 1941.

Dear Dr. Hunter,

The Committee of the Medical War Relief Fund, at a recent meeting, received with great delight the news of the promised donation of £4,500 (Australian) contributed by members of the Association in your country, and sent you by cable a brief message of gratitude.

I find it difficult to express adequately our appreciation of this splendid help. We have not forgotten the very kind offer of our Australian colleagues to care for doctors' children evacuated from Britain, and this fresh evidence of their sympathy and their desire to render assistance of a very practical kind is most gratifying to us and will give great pleasure to the whole profession in this country.

At the meeting to which I have referred, the Committee considered a detailed report on the first year's work of the Fund, and an abridged version of this will shortly appear in *The British Medical Journal*. I hope that our members in Australia, when they see this report and read the typical case summaries which it contains, will feel satisfied that they have subscribed to a very worthy cause and that the money which they have so generously contributed will be well spent.

The cheque from Australia will carry us considerably further along the road towards our first objective of £50,000. We have a large balance in hand, and although we must expect the demands on the Fund to be much heavier in the

future than they have been hitherto, the response to our appeal encourages us to believe that the profession will not fail to provide the necessary assistance in every deserving case, whatever the extent of the need may ultimately prove to be.

Please make it known that the magnificent contribution from our Australian friends gives us particular pleasure, and convey to all concerned our cordial greetings and sincere thanks.

Yours sincerely,

(Sgd.) H. S. SOUTTAR,

Chairman,

Medical War Relief Fund Committee.

The Royal Australasian College of Physicians.

A REPORT ON MEDICAL SOCIOLOGY.

THE following is a summary of a report by Professor John Bostock, of Brisbane, to the Royal Australasian College of Physicians. The report was considered at a joint meeting of the councils of the Royal Australasian College of Physicians and the Royal Australasian College of Surgeons and then forwarded to the Federal Council of the British Medical Association in Australia. The Federal Council sent it to the several Branches.

It is an inescapable fact that much of the world chaos is due to indifference by great masses of citizens as to the principles underlying a sound sociological system. They have been content to interpret a citizen's duty as the casting of a vote at triennial elections. Often even this is regarded as a nuisance. To a great extent the growth of specialization has conduced to this state of affairs, which leaves humanity at the mercy of such tyrants as Hitler and Mussolini.

Specialization has assisted by keeping men's noses so ground to the millstone of their particular work that they develop a myopia as to their surroundings. Of no section of the community is this more true than that of the medical profession. Whilst here and there enthusiastic doctors have sacrificed a little pecuniary gain on the altars of social service, the vast majority take no interest in sociology beyond that of critic after the event. This attitude of mind is the more harmful and reprehensible because medicine is not merely a learned profession, but it is one which impinges upon every phase of human endeavour. There is hardly a sociological problem which does not need our medical service to solve at least a moiety of its difficulties. Recognizing this, our profession should realize its civic obligation and take an enthusiastic and untiring action on all social problems. Every State and Federal government, local council, progress and reform body should have a doctor in its midst. In addition, our profession should be clament in the field of active health propaganda, whether by wireless, Press or other avenue.

The Royal Australasian College of Physicians is to be congratulated on its public-spirited work towards the betterment of medical education. It aims to raise the standard of medical practice. Its ranks are recruited from the most intelligent and honoured members of the profession. The time has arrived for us to ask a searching question. As educators of our profession are we doing enough in the broad field of civics? Do we realize that it is tragic for us to see birth control produce race suicide, doles create mass degeneracy, misapplied national psychology foster recurring wars, ill-directed pensions produce pauperism on the one hand and intolerable financial burdens on the other *et cetera*? Yet we do nothing as a body either to probe the causes or find a solution. Strictly speaking, these are political questions, but have the test tube and medical gadgets so fettered our souls that we have no time to spare for that great humanitarian quest which illumed the souls of the old physicians?

In our opinion the time has arrived for the Royal Australasian College of Physicians not merely to take cognizance of its civic responsibility, but to take energetic steps in helping to mould public opinion. We make the following suggestions:

1. The council should encourage the formation of sociological study groups throughout the States. Whilst many other problems will present themselves, the following may be taken as examples: medical representation on civic bodies; the approaching population stalemate—causes and remedies; post-war reconstruction—medical services *et cetera*; federation of democracies—its medical implications; betterment of international relationships (for example, Australian-American associations *et cetera*); League of Nations health services; socialization of medicine pros and cons; the psychological causes of war and their remedy; the drive to victory; the war pension problem *et cetera*; educational curricula of school children (for example, place of eugenics, mothercraft, handicrafts, examinations *et cetera*).

2. The college must arrange for publicity by journals, newspapers and radio. Such publicity under its ægis would not infringe the ethics of medical advertising.

It is conceivable that the cost of such services would militate against their acceptance. Our view is that in this time of national emergency, when the very existence of our democracy is at stake, we must economize on formal expenditure to commence the greater work which concerns the core of our national and international life.

Special Correspondence.

NEW ZEALAND LETTER.

BY OUR SPECIAL CORRESPONDENT.

War in the Pacific.

THE wave of public interest aroused by the inauguration of the General Practitioner Service in New Zealand under Social Security in November, 1941, had scarcely subsided when war came closer and has effectively swamped medical matters in the public mind

General mobilization of military reserves, plans for evacuation of coastal towns, raid precautions in the cities, are all-important. The drafting of many more practitioners to military camps for an indefinite period has placed a very heavy burden on those that remain. It would seem that organized arrangements for night work, if not for the whole twenty-four hours, will soon be necessary if civilian services are to be maintained.

Similarly, public hospitals are feeling the strain of depleted staffs and increased demands. Although great inconveniences and curtailment of services have already been imposed, no major rearrangements have yet been made.

There have been important additions to the drug tariff under the pharmaceutical benefits of Social Security, dated January, 1942. Liver products for injection, desiccated stomach tissue, sulphonamide with its salts, compounds and derivatives, and other substances are now included. Simultaneously with this has come an official request for strictness in prescribing in general, in order to conserve limited stocks of drugs in the country.

With regard to the modes of collection of the fees payable under medical benefits to the doctors, the official British Medical Association attitude—not without criticism from within—remains as before. The patient pays the doctor his ordinary fee, and on the basis of his receipt goes to the post office to claim his seven shillings and sixpence drawback. The Minister of Health announces that many doctors are using the simpler and more economical way of collecting directly from the Government, whether they ask for an additional three shillings a time or not.

X-ray diagnostic services, free at public hospitals, have been promised to be available from private radiologists, the fund paying a proportion of the charges. Though it is said that all details have been agreed upon, the service is not yet promulgated.

Obituary.

Kenneth Mackenzie, M.D., Ch.M., F.R.C.S. (England), F.R.A.C.S., died at the age of fifty-six on January 15, 1942, in Auckland. A son of Sir Thomas Mackenzie, former Prime Minister of New Zealand and High Commissioner in London, he had practised as a surgeon and gynæcologist in Auckland since 1914. He held many posts in medical and public life. Surgeon to Auckland Hospital for twenty-eight years, he will be remembered chiefly for clinical teaching to final-year students, examining at Otago University, and for his work with the Cancer Consultation Committee, and particularly in respect of carcinoma of the *cervix uteri*. He was a former President of Auckland University College Council and of the Rotary Club. His brother Dr. Bruce Mackenzie practises as a radiologist in Auckland, and he leaves a widow and son and three daughters. His passing is greatly regretted in Auckland.

Correspondence.

A NATIONAL MEDICAL SERVICE.

SIR: By now every member will have received a copy of the draft of the proposed revision of our lodge agreements, submitted to us by the Federal Council, and I would appeal to every forward-thinking member to bestir himself to protest against its ratification in its present form.

The scheme propounded by our Federal Council for a national medical service paid lip service to the principle of utilizing general practitioners for health-building and prophylactic service of our people; but what chance will they have of securing national adoption of these reforms if they omit their introduction in some practical form when a revision of our lodge agreements is formulated by us for the acceptance of the lodges, who will later on become the approved societies?

We are being led in this matter, just as was our profession in the Old Country before the *National Insurance Act* was passed, and unless we and our leaders act promptly, we shall be "guided" into the same stupid and humiliating position, so injurious to medical prestige in England in 1912.

There is no doubt that this is an excellent instrument if its purpose is (as it was described by a high official in our Branch) "to provide a G.P. service for minor ailments for low income people".

It is my claim, however, that our function as general practitioners is to prepare for their work of reproduction bodies whose health has been maintained in a sound state from their infancy, by dealing with deficiencies and correcting physiological faults.

Science, our own medical science, has already brought the prediction and prevention of pathological changes within the realms of practical politics, and we are bound by our Hippocratic oath to give our best to our people irrespective of their financial ability to pay our full fees or not. But on the mere grounds of self-preservation I contend we are taking a perilous course if we withhold our best at such a time of national and racial emergency.

These are days of widespread publicity, and the world at large is rapidly becming aware that "until we do these things . . . we are just touching on the veriest fringe of the best practice of medicine", as has been stated so trenchantly by Dr. Thomson in his letter on "A General Medical Service for Australia" in your number of January 17, 1942.

During our discussions here I proposed that:

> Our contracts should embody, as a condition of membership in every medical scheme, that the members and their dependants either submit themselves for periodical medical examinations or pay a higher premium for their treatment when ill;
>
> Infants up to 1 year, every month;
>
> Children from 1 year to adolescence, four times a year;
>
> The parents, twice each year (up to age forty).

Lodge agreement revision provides a most valuable opportunity for us to show that we are determined not to perpetuate the existing method of awaiting the arrival of disease and the institution of pathological changes before the doctor is called in.

It gives us the chance to introduce a matter closely concerned with the health of our contract patients into our discussions, as well as matters of our pay and our conditions of service, and would establish a precedent of the utmost importance for subsequent national negotiations.

Not to introduce this reform, so vital to the public health, would be nothing less than a betrayal both of our own personal and professional interests, and those of our public as represented by our contract patients.

Yours, etc.,

STANLEY BOYD.

Gnowangerup,
Western Australia,
January 25, 1942.

ATTEMPTED SUICIDE.

SIR: The conclusions drawn by Dr. McGeorge in his analysis of 523 cases of attempted suicide differ so much from the accepted facts about suicide that some explanation should be sought for.

The facts about suicide are based on statistics from many countries for over fifty years. As the number of suicides so investigated has been vast and the margin of possible statistical error negligible, the statistical conclusions so obtained must be accepted as the true facts about suicide.

The number of women suiciding to 100 men varies from 25 to 33 in different countries. In Australia the figure is around 27. Dr. McGeorge's series consists of 244 women and 279 men, or 87 women to 100 men. The figure of 87 is so high that one wonders whether the women in the series were grossly inefficient in their attempted suicide or that they did not really mean to suicide.

The risk of suicide progressively increases from youth to old age in the case of men, and in females from youth to about forty, when the risk remains fairly constant until old age. If the attempted suicide rate in Dr. McGeorge's series per 100,000 of the population in New South Wales is calculated (the calculations here are based on the figures given in the "Official Year Book of New South Wales", 1934-1935, pages 657-658) the following table results:

Age Group.	Attempted Suicide Rate.
15-19	17
20-29	31
30-39	29
40-49	32
50-59	35
60-69	23
70 and over	12

In other words, the attempted suicide rate remains fairly constant from 20 to 59 and it then decreases sharply. Whereas in reality not only should there be a progressive increase from 20 to 59, but there should also be a progressive and fairly steep rise after the age of 60.

The methods, too, adopted by Dr. McGeorge's patients give rise to the suspicion that many of the patients had no intention of suiciding. The "Official Year Book of New South Wales" (*opere citato*, page 718) gives a summary of the methods used per 100 suicides from 1931-1935 inclusive: poison 28, shooting 24, cutting 12, hanging 11, gas 11, drowning 7. In Dr. McGeorge's series the following percentages of methods were used: poison 36 (to the nearest figure), cutting 21, precipitation from height 12, drowning 6, hanging 4, firearms 2. Now whilst hanging and shooting together account for 35% of the successful suicides, they account for only 6% of the unsuccessful ones. Similarly, whilst only 12% of persons suicided by cutting, 21% of the persons in this series tried to do so. But the greatest contrast of all is to be found in suicidal attempts by precipitation from a height. This method is not mentioned in the official New South Wales figures and is very low in the English figures quoted in this series, and yet it accounts for nearly 12% of all attempted suicides given by Dr. McGeorge.

The fact is clear that the percentage of attempted suicides in this series by cutting and precipitation from a height is far too high, whilst those by firearms and hanging are far too low. The conclusion must be forced on us that the patients in the series as a rule avoided methods, such as shooting and hanging, which are dangerous to life, and adopted methods which, whilst dramatic and convincing in themselves, leave wide open the gate to recovery. Superficial cuts on the wrists and throat may be a gesture of defiance to relatives and to life in general without necessarily constituting a real suicidal attempt. To be rescued on the brink of The Gap or to be prevented in time from a jump off a hospital veranda is a drama so often brilliantly acted by one who has to adopt unorthodox methods to enforce her will on unwilling relatives.

Similarly, the finding that the fewest suicides occur in the spring is the exact opposite to the real truth. Again, the statement that "labourers head the list . . . as might be expected, for the unskilled labourer finds life harder than one who has a trade or profession to make employment more certain" is not only inconsistent with the statement on page 71, "it is worthy of note that unemployment is last on the list" (that is, of the causes of attempted suicide), but also with the real facts. Figures prove that those most prone to suicide are professional and business men, including doctors and legal men, men of independent means and the like. Labourers are well down on the list. It is not only an assertion, but a truth, "that suicide is at its highest level during times of financial depression". This is well illustrated by the very high suicidal levels during the 1896 and 1930 financial depressions in Australia.

It is true that unemployment is not a high precipitating cause of suicide in labourers because they have so little to lose. They have little when working and when unemployed they have the dole and the charity of their neighbours to help them. On the other hand professional and business men, who are apt to set too high a value on worldly

possessions, are in danger of losing too much during a financial depression. They and not the labourers swell the suicide rate during financial depressions. Poverty in itself is not a prolific cause of suicide, but the descent from affluence to poverty is.

The criterion adopted by Dr. McGeorge that "suicides from the psychiatric point of view fall into two groups, the sane and the insane", raises the question of how are we going to define the borderline between the two. Numerous authors in the past have tried to separate the sane from the insane among persons suiciding, and their results range from 20% to 70% of insane persons amongst those suiciding. The criterion may be all right in the obvious cases of sanity and insanity, but when we see the most eminent psychiatrists differing in the witness box about the mental state of particular patients, who is to decide dogmatically whether a particular patient should be classified in the sane group or the insane one? "Who shall decide when doctors disagree?" No, sir, the criterion adopted by Dr. McGeorge is far too nebulous for the borderline cases.

The conclusions thus drawn by Dr. McGeorge are so fundamentally different from the accepted facts about suicide that one is driven to the conclusion that persons suiciding and those attempting it are essentially different. This is borne out by clinical observation. Patients who are determined to suicide seek the most lethal methods of self-destruction and take care that their preparations are secret. Their attempt is usually so successful that it is exceptional to see them in mental hospitals or reception houses. Even if their attempt is unsuccessful they retain their determination to suicide for a time, and in mental hospitals they often elude the continuous observation insisted upon by Dr. McGeorge and suicide. On such patients the statistics of suicide are based.

On the other hand, series of attempted suicide include a majority of patients who do not really intend to suicide. They avoid the most lethal methods of self-destruction, often have an audience for the attempt or at least they pick a site where they can easily call attention to themselves. Having defied the elements like Ajax, they are satisfied. No matter how depressed such patients may be, they do not require continuous observation, a fact I have repeatedly demonstrated during the past four years.

It is quite obvious that conclusions drawn from a series of attempted suicides cannot be validly applied to suicides in general. If Dr. McGeorge had realized this fact, his despairing note that he had no suggestion with regard to prophylaxis, a statement which is so contrary to historical, religious, racial and statistical evidence, could never have been written. Suicides like the poor have always been, but their incidence, whether high or low, depends on whether men worship God and their country and not themselves. If they are prepared to sacrifice religion and patriotism for their own selfish interests, then the suicide rate is high. If they are truly patriotic and religious, then the suicide rate is low. The increasing suicide rate in recent years, coupled with a falling birth rate, is so characteristic of the development of selfishness that truly patriotic Englishmen have trembled for the safety of the Empire. With the awful example of France, with its high suicide rate and low birth rate, before our eyes, we can still see English and Australian doctors preaching euthanasia, limitation of families and increasing indications for abortion. In all their discussions one looks in vain for the note that the interests of the country are paramount and that the interests of the individual are but secondary.

The pessimism of Dr. McGeorge extends not only to worldly things, but also to the conceivable discontent of some of the inhabitants of the Elysian fields. He has evolved a new theology, which not only contradicts the very word "Elysium", but is a direct negative of the perfect happiness to be found in the heaven not only of the Christian and Jewish religions, but of many pagan ones as well. This doubting of spiritual values has been a feature of materialistic philosophers since the time of Zeno and his followers in ancient Greece. Wide acceptance of such views has thrown men into the gloomy introspection of their own pessimistic thoughts, unrelieved by the expectation of something higher after death. Their life became unbearable to them and many of them suicided. It seems most strange and inconceivable that even the suspicion of such a doctrine, which has precipitated so many suicides in the past, should find its way into a paper which seeks to not only understand but to draw apparent conclusions from a series of patients who attempted suicide. That Dr. McGeorge himself does not believe in his own statement that "no suggestion can be made with regard to prophylaxis" is his acceptance of the views of the authorities he quotes to show the influence of religion in the prevention of suicide. Such contradictions can only suggest that the final conclusion drawn from the analysis of the 523 cases of attempted suicide is more of a literary effort than an analysis of known facts.

Yours, etc.,

S. J. MINOGUE.

The Hospital,
Kenmore,
New South Wales.
January 26, 1942.

THE TREATMENT OF BURNS.

SIR: Dr. T. E. Wilson, in his article in THE MEDICAL JOURNAL OF AUSTRALIA of January 31, makes a slip of the pen when he refers to a 5% solution of electrolytic hypochlorites.

This direction might lead to undesirable effects if followed literally.

The correction is to be found in Bunyan's article in *The British Medical Journal* of July 5, 1941, page 3, where he says: ". . . so that when we talk of a 5% solution of E.S.H. we mean 1 part of 1% electrolytic sodium hypochlorite to 20 parts of water".

Yours, etc.,

CRAWFORD MCKELLAR.

143, Macquarie Street,
Sydney,
February 2, 1942.

MAN POWER.

SIR: The letter of Dr. Davis necessitates reference to facts. The system of investigation by standing medical boards saved the British Government large sums in pensions because clinical evidence was recorded at the time of examination and consequently there was no dispute afterwards.

I do not trust to memory, as Dr. Davis suggests, but embodied the result of the work in a book of mine, "A Vision of the Possible", in which the enormous gain in efficient man power is set out in precise terms.

Without the system it is doubtful whether Field Marshal Allenby would have succeeded in Palestine.

Finally, the observation quoted, of sending a man to work if suffering from a disease which would kill him in six months' time, was not mine, but that of the medical general under whom I served, and subject to such a man's ability to do such work, as he put it, I entirely agree with it in a national emergency.

As regards complaints, which were very few, they were dealt with in decisive fashion after investigation by the commander-in-chief, as they should be anywhere.

Yours, etc.,

JAMES W. BARRETT.

105-109, Collins Street,
Melbourne,
February 2, 1942.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 28, of January 29, 1942.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Reserve.

Termination of Appointment.—The appointment of Edward Vanderbyl Waddy Pockley as Surgeon Lieutenant is terminated, dated 4th January, 1942.

AUSTRALIAN IMPERIAL FORCE.

Australian Army Medical Corps.

The following officers are seconded from the date shown: Lieutenant-Colonel N. H. W. Saxby and relinquishes command of a Field Ambulance, and Major C. R. Blomfield, 5th November, 1941.

Captain G. G. Cooley is transferred from Reinforcements, 28th August, 1941.

Major (Temporary Lieutenant-Colonel) H. M. Fisher is appointed to command a Field Ambulance, *vice* Lieutenant-Colonel R. H. Russell, D.S.O., 10th November, 1941.

Major (Temporary Lieutenant-Colonel) J. M. Dwyer is appointed to command a Field Ambulance, *vice* Lieutenant-Colonel N. H. W. Saxby, 10th November, 1941.

The undermentioned officers are transferred from Reinforcements from the dates shown: Captains M. H. Colyer, A. D. Reid, R. de G. Burnard, J. Z. Huie, H. J. Delohery, S. E. Francis, L. Abramovitch, K. S. Wallace, R. B. Lynch, R. S. Bennett, S. C. Barrett, K. J. Grice, V. M. Putland, M. M. Perl, E. Clarke, R. P. Carew-Smyth, W. J. Skinner, W. Gove, E. H. S. Mancy, D. O. Crompton, H. J. Edelman, D. L. G. Thomas, W. K. Manning, T. M. Clouston, N. F. Laidlaw, R. Hertzberg, N. Joseph and J. I. Hayward, 3rd September, 1941, L. P. Gray and H. C. Mulcahy, 7th September, 1941, and J. L. D. Scott, 29th September, 1941.

To be Colonel.—Lieutenant-Colonel D. C. Pigdon, E.D., and relinquishes command of an Australian Convalescent Depot, and is appointed to command an Australian General Hospital, 1st August, 1941 (in lieu of the notification respecting this officer which appeared in Executive Minute No. 115/1941, promulgated in *Commonwealth Gazette* No. 210 of 23rd October, 1941).

To be Lieutenant-Colonels.—Majors R. M. W. Webster, M.C., and is appointed to command an Australian Convalescent Depot, 1st August, 1941 (in lieu of the notification respecting this officer which appeared in Executive Minute No. 115/1941, promulgated in *Commonwealth Gazette* No. 210 of 23rd October, 1941), J. G. G. White, 14th September, 1941, C. H. Osborn, 15th September, 1941, and W. A. Bye, 13th November, 1941.

To be Majors.—Captain R. E. Maffey, 13th October, 1941, C. R. Furner, 14th October, 1941, B. H. Anderson, 15th October, 1941, and K. J. Fagan, 16th October, 1941.

Permanent Supernumerary List.

Captain N. R. Godby is transferred from Australian Army Medical Corps, 22nd October, 1941.

The undermentioned officers are transferred from Australian Army Medical Corps from the dates shown: Lieutenant-Colonels M. L. D. McKeon, 24th October, 1941; L. C. E. Lindon, E. B. Jones and A. S. Walker, 6th November, 1941; and Captain H. G. Prest, 7th November, 1941.—(Ex. Min. No. 14—Approved 28th January, 1942.)

AUSTRALIAN MILITARY FORCES.

AUSTRALIAN ARMY MEDICAL CORPS.

Seventh Military District.

Captain (provisionally) A. J. M. Nelson is transferred from Australian Army Medical Corps, 3rd Military District, with regimental seniority in accordance with his Army seniority, 24th December, 1941.

Captain (Temporary Major) A. D. Frost is transferred from Australian Army Medical Corps, 2nd Military District, and retains the temporary rank of Major, 22nd December, 1941.

The notification respecting the transfer of Major N. W. Markwell, which appeared in Executive Minute No. 121/1941, promulgated in *Commonwealth Gazette* No. 136 of 1941, is withdrawn.

Eighth Military District.

To be Captain (provisionally).—Noel Vernon McKenna, 22nd December, 1941.

Northern Command.

First Military District.

Captain (Temporary Major) R. A. Maxwell relinquishes the temporary rank of Major and is transferred to the Reserve of Officers (A.A.M.C.) and to be Honorary Major, 30th November, 1941.

Honorary Captain R. B. Salter is appointed from the Reserve of Officers (A.A.M.C.) and to be Captain (provisionally), 21st November, 1941.

To be Captain (provisionally).—Frank Hatcher, 22nd December, 1941.

To be Honorary Captain.—Benjamin Hooper, 21st December, 1941.

The following officers are appointed from the Reserve of Officers, and to be Captains (provisionally): Honorary Captains B. N. Adsett and T. R. Neville, 5th December, 1941; P. Black, 9th December, 1941; and W. Arrowsmith, 12th December, 1941.

To be Honorary Captain.—Paul Francis Hyndes, 24th December, 1941.

Captain (provisionally) D. A. Dowling is transferred to the Reserve of Officers (A.A.M.C.), 11th December, 1941.

The following officers are appointed from the Reserve of Officers, and to be Captains (provisionally): Honorary Captains J. A. Emmett, H. C. Murphy and A. R. Townsend, 9th December, 1941, J. V. Hynes, J. W. Woodburn and T. K. Durbridge, 11th December, 1941, P. Black, J. L. Dart and H. W. A. Forbes, 12th December, 1941, and K. B. Shallard, 12th January, 1942.

To be Majors (temporarily).—Captain D. B. Walker and Captains (provisionally) T. M. Mansfield and D. F. Millar, 2nd January, 1942.

The resignation of Honorary Captain C. W. Kingston of his commission is accepted, 8th December, 1941.

To be Honorary Captain.—David Paul Hannaford Schafer, 26th December, 1941.

Eastern Command.

Second Military District.

Honorary Major F. S. Hansman is appointed from the Reserve of Officers (A.A.M.C.) and to be Captain (provisionally) and retains the honorary rank of Major, 13th November, 1941.

To be Major (temporarily).—Captain (provisionally) C. O. Donovan, 15th December, 1941.

The name "John Maloney" which appeared in Executive Minute No. 220/1941, promulgated in *Commonwealth Gazette*, No. 226 of 1941, is amended to read "John Moloney".

To be Honorary Captains.—Harold Eldon Moran, Adrian Gustave Nelson Basser and Raymond Maxwell Glennie Holmes, 15th December, 1941, Gerald Field and Paul Alexander Sheehan, 17th December, 1941, and John Northleigh Carter, 19th December, 1941.

The provisional appointment of Captain H. M. Hollingworth is confirmed.

Honorary Captain I. D. R. Gardiner is appointed from the Reserve of Officers (A.A.M.C.) and to be Captain (provisionally), 28th November, 1941.

To be Lieutenant-Colonel (temporarily).—Captain (Temporary Major) S. D. Meares, 1st January, 1942.

To be Captains (provisionally).—Rupert Sheldon, John Patrick Ryan and Richard Daniel Condon, 3rd January, 1942.

To be Honorary Captains.—Ronald Maxwell Rawle, Roy Warren Russell-Jones, 19th December, 1941, and Alan William Hale Bailey, 22nd December, 1941.

Captain A. F. Smith is transferred from Australian Army Medical Corps, 7th Military District, 10th November, 1941.

Captain (provisionally) (Temporary Major) N108414 P. Tillett is transferred from Australian Army Medical Corps, 7th Military District, and retains the temporary rank of Major, 25th November, 1941, and is retired, 19th December, 1941.

The following officers are appointed from the Reserve of Officers (A.A.M.C.): Major N99319 G. R. Halloran, 26th October, 1940, Captains (provisionally) N289710 H. I. McKenzie, 20th October, 1941, N281823 W. G. H. Epps, 27th October, 1941, Captains C. R. Alexander, 27th November, 1941, and A. M. Welsh, 16th December, 1941, and Captain (provisionally) N281460 T. Wunderlich, 18th December, 1941.

The following officers are appointed from the Reserve of Officers (A.A.M.C.) and to be Captains (provisionally): Honorary Captains N108185 D. A. Selby, 4th April, 1941, N280887 J. C. McInerney, 1st October, 1941, N279444 K. P. Clifford, 6th October, 1941, N281652 B. R. Schloeffel, 22nd October, 1941, L. Sender, 10th November, 1941, J. H. Carman, 14th November, 1941, and N279306 M. J. J. O'Reilly, 18th November, 1941.

Honorary Captain W. H. C. Hughes is retired, 30th December, 1941.

To be Honorary Captain.—Nicholas Campbell Wright, 24th December, 1941.

Southern Command.

Third Military District.

The provisional appointment of Captain C. E. Willing is confirmed, 30th October, 1941.

The resignation of Captain (provisionally) G. F. Salter of his commission is accepted, 8th October, 1941.

Captain (provisionally) J. W. Perry is retired, 2nd December, 1941.

Captain C. E. Willing is transferred to the Reserve of Officers (A.A.M.C.), 1st November, 1941.

The resignations of Honorary Captains C. R. Laing and H. R. Clegg of their commissions are accepted, 28th October, 1941, and 1st December, 1941, respectively.

To be Major (temporarily).—Captain (provisionally) V29260 M. O. K. Hughes, 24th December, 1941.

To be Honorary Captain.—Joseph Bornstein, 29th December, 1941.

The following officers are appointed from the Reserve of Officers and to be Captains (provisionally): Honorary Captains V9377 W. Pook, 12th December, 1940, H. J. Enniss, 4th December, 1941, and D. A. Kidd, 17th December, 1941.

To be Majors (temporarily).—Captain (provisionally) V9377 W. Pook, 24th December, 1941, Captains V113147 V. C. Brown, M.C., 26th December, 1941, and V113245 H. R. Smith, 3rd January, 1942, and Captains (provisionally) V31301 G. J. Loughran and V11237 R. D. Smith, 3rd January, 1942, and 6th January, 1942, respectively.

To be Captain (provisionally).—Raymond Victor Chong, 6th January, 1942.

Fourth Military District.

To be Honorary Captain.—Raymond Wilbur Louis Crosby 24th December, 1941.

Western Command.

Fifth Military District.

Captain (provisionally) H. J. Rowe is transferred from Australian Army Medical Corps, 1st Military District, 25th November, 1941.

To be Major (temporarily).—Captain (provisionally) A. A. Hill, 1st January, 1942.

Honorary Captain W. F. Tomlinson is appointed from the Reserve of Officers and to be Captain (provisionally), 23rd December, 1941.

To be Major (temporarily).—Captain (provisionally) W16093 A. M. Clarke, 30th December, 1941.

To be Honorary Captain.—Robert Percival Hodgson, 2nd January, 1942.

National Emergency Measures.

MEDICAL EQUIPMENT.

THE Medical Equipment Control Committee desires to direct the attention of all members of the medical profession to the following information.

Glycerine.

The following is a letter from the Ministry of Munitions.

Dear Sir,

With reference to the recent restrictions on the sale of glycerine under the National Security Regulations, you are advised that supplies of glycerine for pharmaceutical purposes (Group B Glycerine Regulations) are to be rationed on and from 1st February, 1942, in accordance with the following schedule:

1. Glycerine in aqueous solution or pure, may not be sold without production of a prescription from a medical practitioner.
2. Existing stocks of glycerine packed for retail sale may be disposed of.
3. Pharmaceutical and manufacturing chemists must forward a declaration to their supplier stating that all glycerine purchased by them will be used entirely in the preparation of compounds required for medical purposes.
4. Wholesale druggists are required to furnish with all orders placed with the refiners a declaration stating that the glycerine will be used or re-sold for medical purposes only. They should also keep a record showing the date, amount and name of the purchaser, which must be forwarded to the Controller of Materials Supply on the first day of each month.
5. The use of glycerine in all toilet preparations other than vanishing cream, shaving soap, or shaving cream is prohibited.

Yours faithfully,

H. C. GREEN,
Controller, Materials Supply.

The Department of Supply and Development requests publication of the following information.

Use of Surgical Gauze.

The Medical Equipment Control Committee has had its attention directed to the fact that surgical gauze, bandage cloth, and butter muslin are still being used for non-medical purposes, contrary to provisions of the Medical Equipment Control order issued on January 12.

It has been reported that surgical gauze and butter muslin are being purchased and pasted on windows as a precaution against shattering of glass in the event of air raids. Use of these commodities in this way is an offence against the *National Security Act*. Members of the public are therefore warned that they must comply strictly with provisions of the order, which is designed to conserve very limited stocks of these materials now available.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Bligh, Richard Geoffrey, M.B., 1941 (Univ. Sydney), 241, Miller Street, North Sydney.
Bonnette, Ronald Howard, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.
Brand, Nugent Elliot, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Brown, David Arthur, M.B., B.S., 1941 (Univ. Sydney), Ryde District Soldiers' Memorial Hospital, Eastwood.
Busby, Hugh, M.B., B.S., 1941 (Univ. Sydney), St. George District Hospital, Kogarah.
Byers, Kevin John, M.B., 1941 (Univ. Sydney), Lewisham Hospital, Lewisham.
Byrne, Ross, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Cahill, Arthur Paul, M.B., B.S., 1941 (Univ. Sydney), 91, Old South Head Road, Bondi Junction.
Carey, Harvey McKay, M.B., B.S., 1941 (Univ. Sydney), Eastern Suburbs Hospital, Bondi Junction.
Chesher, Rupert George, M.B., 1941 (Univ. Sydney), Parramatta District Hospital, Parramatta.
Chipperfield, Margaret Joan, M.B., B.S., 1941 (Univ. Sydney), Renwick Hospital for Infants, Summer Hill.
Church, Louis Vivian, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.
Clements, Alan Twyman, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Conneely, Thomas Gerard, M.B., B.S., 1941 (Univ. Sydney), Mater Misericordiæ Hospital, Crow's Nest.
Cumberland, Victor Henry, M.B., B.S., 1941 (Univ. Sydney, 19, Gillis Street, Wollstonecraft.
Dalton, William Redmond, M.B., 1941 (Univ. Sydney), Lewisham Hospital, Lewisham.
Day, Prudence Ursula, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Dey, David Lindsay, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Diethelm, Anton Emil, M.B., B.S., 1941 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Dolman, Bruce Hunter, M.B., B.S., 1941 (Univ. Sydney), Saint George District Hospital, Kogarah.
Dynon, Geoffrey Patrick, M.B., B.S., 1941 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Edwards, Roger Henry, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Emdur, Hyman, M.B., B.S., 1941 (Univ. Sydney), 31, Hollywood Avenue, Bondi Junction.
Filshie, Ian, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Fogarty, Diarmuid, M.B., B.S., 1941 (Univ. Sydney), Lewisham Hospital, Lewisham.
Fraser, William Hector Munro, M.B., B.S., 1941 (Univ. Sydney), Mater Misericordiæ Hospital, Crow's Nest.
Gillogley, Jack Francis, M.B., B.S., 1941 (Univ. Sydney), Marrickville District Hospital, Marrickville.
Greenwell, Peter Howard, M.B., B.S., 1941 (Univ. Sydney), 5, Locksley Street, Killara.
Hambly, Colin Keith, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.
Hammond, Brian Thomas, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.
Harrison, Colin Patrick, M.B., B.S., 1941 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Hartley Smith, Frances, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Hegarty, Vincent Henry, M.B., 1941 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Hellestrand, Alan Lacey, M.B., B.S., 1941 (Univ. Sydney), St. George District Hospital, Kogarah.
Hellestrand, Jean Marian, M.B., B.S., 1941 (Univ. Sydney), Rachel Forster Hospital, Redfern.
Hillyer, William, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Hogan, Allan Barry, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.
Holman, Elton Dudley, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Holmes, Roland Adrian Glennie, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Jack, Ian Bruce, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.
Johns, Kevin, M.B., B.S., 1941 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Joseph, Lynn Harvey, M.B., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Keller, Walter Lloyd Holcombe, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Kent, Merrian Winifred, M.B., B.S., 1941 (Univ. Sydney), Royal Hospital for Women, Paddington.
Khan, Edward Joseph, M.B., 1941 (Univ. Sydney), 156, Chalmers Street, Sydney.
Kidd, Donald Stuart, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.
Kiely, Roger John, M.B., 1941 (Univ. Sydney), Balmain and District Hospital, Balmain.
King, Keith John McInnes, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Lane, John Charles, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.
Lee, Ernest William, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.
Lundie, Arthur James, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Macintosh, Isobel Patricia, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Macleod, Kenneth William, M.B., 1941 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
McNeil, Angus, M.B., B.S., 1941 (Univ. Sydney), Balmain and District Hospital, Balmain.
Malouf, Joseph Michael, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Manery, Mary, M.B., B.S., 1941 (Univ. Sydney), Prince Henry Hospital, Little Bay.
Mason-Johnson, Jean, M.B., B.S., 1941 (Univ. Sydney), Prince Henry Hospital, Little Bay.
Mayer, Jessie Findlay, M.B., B.S., 1941 (Univ. Sydney), St. George District Hospital, Kogarah.
Merchant, Lance Verdun, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Miller, George Joseph, M.B., B.S., 1941 (Univ. Sydney), Mater Misericordiæ Hospital, Crow's Nest.
Moloney, John, M.B., B.S., 1941 (Univ. Sydney), Prince Henry Hospital, Little Bay.
Mooney, Bede Patrick Francis, M.B., B.S., 1941 (Univ. Sydney), Mater Misericordiæ Hospital, Crow's Nest.
Mulhearn, Michael Harry Gerald, M.B., B.S., 1941 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Newton, Noel Curtis, M.B., B.S., 1941 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Oakley, Donald Waterhouse, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
O'Keeffe, Eva Florence, M.B., B.S., 1941 (Univ. Sydney), Rachel Forster Hospital, Redfern.
Opie, Richard Cunynghame, M.B., 1941 (Univ. Sydney). Royal Prince Alfred Hospital, Camperdown.
Palmer, Alexander Allan, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.
Pennington, Victor Warren, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.
Peterson, Bruce Henry, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.
Potts, Gordon Cameron, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.
Quinn, William Matthew James, M.B., B.S., 1941 (Univ. Sydney), Balmain and District Hospital, Balmain.
Read, Francis Harold, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.
Reddel, Arthur Edward McKenzie, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Rhydderch, Alun Iorwerth, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Richards, Harold Joseph, M.B., B.S., 1941 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Royle, Harold George, M.B., B.S., 1941 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Rundle, Philip Alan, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Sands, John Robert, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Scott-Young, Norman Reginald, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Shirley, Anthony David, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.
Smyth, John Justin, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Stanley, Patricia Julie, M.B., B.S., 1941 (Univ. Sydney), Saint Joseph's Hospital, Auburn.
Stephenson, Arthur Lavender, M.B., B.S., 1941 (Univ. Sydney), Balmain and District Hospital, Balmain.
Stuckey, Douglas Seavington, M.B., B.S., 1941 (Univ. Sydney), 57, Spit Road, Mosman.
Swirles, Colin Moorcraft, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.
Symons, Cedric York, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Tonkin, John Richard, M.B., B.S., 1941 (Univ. Sydney), Marrickville District Hospital, Marrickville.
Vincent, George Kenneth, M.B., B.S., 1941 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Wadsworth, Joseph Robert, M.B., B.S., 1941 (Univ. Sydney), Lewisham Hospital, Lewisham.
Waters, James Howard Stewart, M.B., B.S., 1941 (Univ. Sydney), Balmain and District Hospital, Balmain.
Waugh, Peter Cedric Phipps, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
White, James McBride, M.B., B.S., 1941 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Wiley, Thomas William, M.B., B.S., 1941 (Univ. Sydney), Ryde District Soldiers' Memorial Hospital, Eastwood.
Williams, Owen Upcott, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Windeyer, John Spencer, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Windsor, Morgan Francis, M.B., B.S., 1941 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Wise, Henry Douglas, M.B., B.S., 1941 (Univ. Sydney), Manly District Hospital, Manly.
Goolden, George Anthony, M.R.C.S. (England), L.R.C.P. (London), 1918, c.o. Cunard White Star Line, 1-7, Bent Street, Sydney.
Allingham, Johnston Morton, M.B., B.S., 1941 (Univ. Sydney), St. George District Hospital, Kogarah.
Blashki, Loris Phillip, M.B., B.S., 1941 (Univ. Sydney), Orange Base Hospital, Orange.
Church, John Campbell, M.B., B.S., 1941 (Univ. Sydney), Newcastle Hospital, Newcastle.
Dick Smith, John Beaumaris, M.B., B.S., 1941 (Univ. Sydney), Brisbane General Hospital, Brisbane.
Gillespie, Robert Eddington Rome, M.B., B.S., 1941 (Univ. Sydney), Newcastle Hospital, Newcastle.
Harrison, Francis William, M.B., B.S., 1941 (Univ. Sydney), Cessnock District Hospital, Cessnock.
Livingston, Jean Flora, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.
Macleod, Sadie Catherine, M.B., B.S., 1941 (Univ. Sydney), Brisbane General Hospital, Brisbane.
Murray, Victor Reginald Ernest, M.B., B.S., 1941 (Univ. Sydney), Balranald.
Paul, James Adrian, M.B., B.S., 1941 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Segal, Joseph, M.B., B.S., 1941 (Univ. Sydney), Newcastle Hospital, Newcastle.
Jeffrey, Alan Charles, M.B., B.S., 1941 (Univ. Melbourne), Albury District Hospital, Albury.
McMeekin, Hedley James Parker, M.B., B.S., 1940 (Univ. Melbourne), Broken Hill and District Hospital, Broken Hill.
Conquest, James Augustus, M.B., B.S., 1941 (Univ. Melbourne), Lewisham Hospital, Lewisham.
Hill, William Howard, M.B., B.Ch., B.A.O., 1932 (Univ. Belfast), Scarborough.
Frydlanski, Newach Norbert, c.o. Dr. Miles, Sutherland. Registered in accordance with the provisions of section 17A of the *Medical Practitioners Act*, 1938-39.
Hirsz, Jehuda, Ivanhoe Hospital, Ivanhoe. Registered in accordance with the provisions of section 17A of the *Medical Practitioners Act*, 1938-39.
Marshall, Samuel Simon, 725, Rathdown Street, North Carlton, N.4, Victoria. Registered in accordance with the provisions of section 17A of the *Medical Practitioners Act*, 1938-39.
Rosenfeld, Israel, Wallsend District Hospital, Wallsend. Registered in accordance with the provisions of section 17A of the *Medical Practitioners Act*, 1938-39.
Rosenman, David Hersch, c.o. Mr. Firkin, John Street, Wallsend. Registered in accordance with the provisions of section 17A of the *Medical Practitioners Act*, 1938-39.

The following additional qualification has been registered:

Rose, Thomas Frederick, Newcastle (M.B., B.S., 1933, Univ. Sydney, F.R.C.S., England, 1939, F.R.C.S., Edinburgh, 1939, M.R.C.O.G., London, 1939), F.R.A.C.S., 1941.

The following change of name has been registered:

Szput, Maurycy, Bondi (M.D., Univ. Paris, 1936. Registered in accordance with the provisions of section 17A of the *Medical Practitioners Act*, 1938-39), now Stephens, Max.

Obituary.

BERTHA WOOD.

WE regret to announce the death of Dr. Bertha Wood, of Mont Park, Victoria, which occurred on January 14, 1942.

GEORGE ERNEST CLEMONS.

WE regret to announce the death of Dr. George Ernest Clemons, which occurred on February 5, 1942, at Launceston, Tasmania.

CHARLES GOLDSBROUGH ADAMS.

WE regret to announce the death of Dr. Charles Goldsbrough Adams, which occurred on February 3, 1942, at Calcutta, India.

KEVIN JAMES COLLINS.

WE regret to announce the death of Dr. Kevin James Collins, which occurred on February 8, 1942, at Woollahra, New South Wales.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Allingham, Johnston Morton, M.B., B.S., 1941 (Univ. Sydney), St. George District Hospital, Kogarah.

Waddy, Nanette Stacy, M.B., B.S., 1941 (Univ. Sydney), Tamworth Base Hospital, Tamworth.

Barry, Lorna, M.B., B.S., 1939 (Univ. Sydney), "Strathmore", Great North Road, Abbotsford.

Medical Appointments.

Dr. Robert Owen Fox has been appointed Officer of Health at Whyalla and Iron Knob, South Australia.

The Governor in Council has made the following appointments to the honorary medical staff of the Royal Adelaide Hospital, Adelaide, South Australia: Honorary Clinical Assistants.—Medical Section: Dr. Alan Henry Finger, Dr. Oswald Bertram Lower, Dr. Frederick George Trevor Turner, Dr. Reginald Nevill Cudmore Bickford, Dr. Josiah Mark Bonnin, Dr. Sydney Letts Dawkins, Dr. Ivan Sandilands Magarey, Dr. Malcolm William Miller, Dr. Donald Keith McKenzie, Dr. John Meavious Pedler, Dr. Geoffrey Ernest Peters, Dr. Christopher Bagot Sangster, Dr. Stanley Liddelow Seymour, Dr. Ronald Keith Stockbridge, Dr. Robert Frank West. Surgical Section: Dr. Noel James Bonnin, Dr. Joseph Ruskin Cornish, Dr. Oscar Westcott Frewin, Dr. Garton Maxwell Hone, Dr. Wallace Wilson Jolly, Dr. Gilbert Edgar Jose, Dr. Thomas Davis Kelly, Dr. Sydney Krantz, Dr. Bruce Ernest Lawrence, Dr. James Davidson Mill, Dr. Owen Meredith Moulden, Dr. Neill Horace Munday, Dr. Alistair Campbell McEachern, Dr. Douglas Gordon McKay, Dr. Leonard James Ternough Pellew, Dr. John Love Steele Scott, Dr. George Herbert Solomon, Dr. Alan Hubert White. Orthopædic Section: Dr. Esmond Frank West, Dr. Norman Stannus Gunning. Gynæcological Section: Dr. Wilbur Francis Joynt, Dr. Bertram Eric Wurm, Dr. Claude Vincent Hallett. Ophthalmological Section: Dr. Thomas Leslie McLarty, Dr. Samuel Pearlman. X-Ray Section: Dr. Bertram Speakman Hanson, Dr. Bronte Creagh Smeaton. Radium Section: Dr. Bertram Speakman Hanson, Dr. William Gilfillan. Dermatological Section: Dr. William Gilfillan, Dr. Leslie Wadmore Linn. Vaccine and Asthma Clinic: Dr. John Edwin Bateman. Physiology Department: Dr. James David Fotheringham, Dr. David Wickham Shepherd. Temporary Honorary Clinical Assistants.—Ophthalmological Section: Dr. Edward Donough Mulvihill Ryan. Out-Patient Department: Dr. Vernon James Odlum, Dr. Neil Oldfield. Honorary Anæsthetists: Dr. Joseph Ruskin Cornish, Dr. Robert Hugo Elix, Dr. Andrew John Hakendorf.

Dr. William Henry Norman Randall has been appointed, pursuant to the provisions of *The Charitable Institutions Management Act of* 1885, of Queensland, a Visiting Medical Officer to the Dalby Hospital for Chronic Diseases and the Dalby Training Farm, Queensland.

Diary for the Month.

FEB. 13.—Queensland Branch, B.M.A.: Council.
FEB. 18.—Western Australian Branch, B.M.A.: Branch.
FEB. 26.—South Australian Branch, B.M.A.: Branch.
FEB. 27.—Queensland Branch, B.M.A.: Council.
FEB. 27.—Tasmanian Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility unless such a notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £1 for Australia and £2 5s. abroad *per annum* payable in advance.